A STUDY ON

KALLADAIPPU

the dissertation submitted by

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under the Guidance of

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CERTIFICATE

This is to certify that this dissertation work on **KALLADAIPPU** has been carried out by **Dr. R. SATHYAVATHY** during the year 2010-2013 in the Post Graduate Department of Maruthuvam, Government Siddha Medical College, Chennai- 600106 under my guidance and supervision in partial fulfillment of regulation laid by **The Tamilnadu Dr. M.G.R. Medical University, Chennai** for the final **M.D. (Siddha) Branch I-MARUTHUVAM** examination to be held in **April 2013**.

This dissertation is a record of original work done and it has not been previously formed the basis for the award of any degree.

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CONTENTS

INTRODUCTION

Health is the chief basis for the development of the ethical, economic, artistic and spiritual sides of man .The wealth of a country depends not merely on its natural resources but also on the vitality of its people.

"பிணியின்மைசெல்வம் விளைவு இன்பம் ஏமம்
அணியென்பநாட்டிற்கு இவ்வவைந்து"

- திருக்குறள்

According to the theory of siddhars, the human body is composed of panjaboothas.The universe is also composed of the same.Human body is microcosmic, the universe is macrocosmic.

அண்டத்தில் உள்ளதே பிண்டம்; பிண்டத்தில்
உள்ளதே அண்டம்
அண்டமும் பிண்டமும் ஒன்றே; அறிந்துதான்
பார்க்கும்போதே.

- சட்டமுனி ஞானம்

Human body is functioning with seven udalthathus and by rhythmic run of three uyirthathus. The alteration in uyirthathus leads to diseased condition.

Food plays a vital role in the rhythmic run of three uyirthathus.

"மருந்தென வேண்டாவாம் யாக்கைக்கு அருந்தியது
அற்றது போற்றி உணின்"

- திருக்குறள்

It says that a man who takes food after knowing the digestion of previous food, never needs to take medicine.

One of the disease manifested by irregular diet is **kalladaippu** (Urolithiasis).

Kalladaippu (Urolithiasis) or the formation of calculi at any level of urinary tract is a common disorder as it has worldwide distribution.Particularly common in some geographic locations such as USA, South Africa, south east Asia. It is estimated that approximately 4% of world population experience renal stone disease. 15% of indian population suffer from kidney stones.

Recurrent stone formation is a common part of the medical care of patients with stone disease.Calcium-containing stones, especially calcium oxalate monohydrade, calcium oxalate dihydrade and basic calcium phosphate are the most commonly occurring ones to an extent of 75-90% followed by magnesium ammonium phosphate (struvite) to an extent of 10-15%, uric acid 3-10% and cysteine 0.5-1% (4-6).

Even though many siddhars explain about this disease, Yugi in hisyugivaidhyachindhamani elaborately described about its aetiology, pathology, classification, clinical features and prognosis of kalladaippu.

The diseases of urinary system are divided into two. They are

"நீரினைஅருக்கல் நோய்"

"நீரினைபெருக்கல் நோய்"

This disease kalladaippu comes under the classification of "நீரினைஅருக்கல் நோய்"

Which is producing low urinary output and driesup the urine and form urinary calculi due to various aetiological factors. The managerial professionals, sedentaryoccupationals are having higher incidents of kalladaippu than the manual workers.

People who run to earn,forget to look after themselves,leads to loss of good health.In this stressful life when they miss to drink water to flush out the waste product leads to calculi disease, kalladaippu.

In practice, many people were afraid of surgery and they like to take herbal medicines which are free from side effects. The patients who are recommended for surgery by urologists also brought stones after taking siddha medicines in our op. So that the author choose **kalladaippu (urolithiasis)** as her dissertation topic to find out a complete cure without surgery.

Forthis, the author select **Megarajangachooranam**. Referred in Athmaratchaamirtham (Pa.no.430) to study its effect on **kalladaippu** pharmacologically, clinically.

AIM AND OBJECTIVES

AIM: The aim of my dissertation work is to evaluate the efficacy of the trial drug, **Megarajangachooranam** both clinically and experimentally in the treatment of **kalladaippu.**

OBJECTIVES

- To collect the authoric measures and literature reviews of kalladaippunoi in ancient siddha and modern literatures.
- To have an idea of the incidence of the disease with regard to age, sex, occupation , socio economic status, food, climatic conditions and precipitating factors etc.
- To expose the efficacy of siddhar's diagnostic principles.
- To utilize the possible methods to confirm the diagnosis and prognosis.
- To have clinical trial on patients with kalladaippunoi with selected siddha medicine.
    - Megarajangachooranam (Athmaratchamirtham)
- To evaluate

  Toxicological screening
    - Acute
    - Sub acute
- Pharmacological screening
    - Lithotriptic
- To find out the statistical analysis of clinical study.

REVIEW OF LITERATURES

SIDDHA ASPECTS

In siddha system, the disease Kalladaippu is mentioned by Yugi Munivar in Yugi vaidhya chinthamani 800. It is one of the Urinary diseases, which comes under Neerinai Arukkal Noi.

"நீரிருவினைக் குணத்தை
நீயறிவித்துக் சொல்வாய்
நீரினைப் பெருக்கலொன்றே
நீரினை யருக்க லொன்று
நீரிழிவுடனே கொல்லும்
நீர்க்கட்டு வினைகளொன்று"

THERAN KARISAL
SIDDHA MARUTHUVAM (POTHU) P.NO.460

1.Verupeyar (Synonyms)

Atchmari Rogam

2.Eyal (Definiition)

Definition of Kalladaippu is mentioned in many Siddha Text Books. Few of them are as follows,

"தானென்ற மூத்திரத்தில் நறநறவென்று
தங்கிதோர் பொடியெனும் மணல்தானப்பா
வானென்ற சிறியதொரு மணல்தானப்பா
வளமாக வந்து விழும் நோய்க்குத் தானே
ஏனென்ற அஸ்மரி ரோகமென்ற பேராம்
எளிதாகக் கல்லுகள் தான் விழுகும் போது
கோனென்ற குணடிக்காய் மூத்திரக்குழலப்பா

குணமான மூத்திரப்பை நீர்தாரை கேளே
கேளடா முன் குறியில் எரிச்சல் கண்டு
கொடியாக வேதனைகள் காட்டுமப்பா
வாளடா சிறியதொரு கற்கள் தானே
வளமான முத்திரப்பை குழல் வழிப்பாயாயத்
தேளடா வரும்போது திரேகந்தன்னில்
தெரிப்பது போல யிருவேதனை செய்யும்பாரு
நாளடா கற்கள் தானிறங்கி விட்டால்
நலமான வேதனைகள் தான் தீரும் பாரே.."

AGATHIYAR GUNAVAGADAM
P.NO.296

Agathiar says the definition of Kalladaippu as sand like crystals found in urine, followed by small size of stones excreted in urine. Stones are stagnated in kidney, ureter, urinary bladder and urethra. Pain with burning sensation starts in urethral orifice followed by agonizing pain when the stone moving in urethral tract from the bladder. The pain is relieved., when the stone is removed or expelled.

Large concentration of urine in the urinary bladder produces calculus or gravel. It causes difficulty in passing urine.

According to Siddha Maruthuvam

Sudden obstruction to the flow of urine, pain in the base of the penis in males and clitoris in females burning micturition, loin to groin pain, passing of small sand like stones along with urine are the cardinal features of this disease.

According to Jeevaratchamirtham

Kalladaippu is defined as pain in and around the umbilicus, fever, dysuria and urine smelling like that of goats urine.

NOI VARUM VAZHI (Aetiology)

The causes of the disease in various siddha text books are,

கலங்கினதோர் தண்ணீர்தான் குடித்தபேர்க்கும்
கல்லெலும்பு மயிர்மண் தான் கலந்தன்னத்தில்
அலங்கினதோரன்ன கலந்தருந்தலாலும்
அழுகலோடு முத்த பண்ட மருந்தலாலும்
மலங்கினதோர் மாப்பண்ட மருந்தலாலும்
மந்தத்தில் வாய்வான பதார்த்தந்தன்னை
துலங்கினதோர் ருசிதன்னிற் சுவைத்தலாலும்
சுருக்காய்க் கல்லடைப்பு வந்து தோன்றுந்தானே

தெளிந்ததோர் கல்லடைப்பு உற்பத்தி கேளாய்
சிறிது நாட்டொடங்கிய மேகந்தன்னில்
தளிந்தோர் சலப்பையிலுதிரத் தோய்ந்து
சந்த சத்தாகவே பருத்துக் கொள்ளும்
வளிந்ததோர் வாத பித்த கோபித்தக்கால்
வந்து பெரு கல்லாய் நீர்வழியடைத்து
நளிந்த்தோர் நாலுவிதத் கல்லடைப்பு
நண்பான வரலாறு நாட்டக் கேளே.

YUGI VAITHYA CHINTHAMANI
PAGE NO.283

This poem says, in chronic Mega noi (Syphilitic disease) the semen will stagnate for a long time, in the urinary tract, so it will obstruct the urine flow. The urine constituents will easily depoit on the urinary tract and form the stone, at that time by vitiation of Vatham and Pitham these small stone become larger in size and, block the urinary passage. Urinary stone are also formed due to the drinking of contaminated hard water, taking of food mixed with sand and small

stones, consuming of contaminated food articles, food containing more carbohydrate, unhealthy food habits etc.

“குருதி மாசுறல் புணர்ச்சி மிகுதல்
சிறுநீ ரடக்கல் விரையில் அடிபடல்
நீரியந் தாக்கல் சிறுநீ ரடக்கல்
வளிநோய் மிகுக்கு முணவும் ஒழுக்கம்
கடைப்பிடித் திடுதல் மேக முதற்பல
பிணியுறல் எனுமிவை யடிப்படை யாகக்
கல்லடைப் பென்னுங் கடும்பிணி விளையும்.”

SIDDHA MARUTHUVANGA SURUKKAM
PAGE NO.142

Derangement of humour in blood, Excessive indulgence in sexual activity or sexual perversion, Trauma on testes, Suppression of urine and semen.

Inflammation of bladders, Syphilis (Mega noil), Stagnation of urine in urinary tract. Dryness of semen causes the formation of stones, Increased intake of food that, cause flatulence.

There are 14 Natural urges in the body. The urine and seman are also the natural urges of our body. So suppression of any one it causes fever, retention of urine which favours urinary calculi, chest pain, arthralgia, and white discharge.

“நீரினை தடுத்தல் செய்யின்
நீர்க்கட்டுத் துவாரம் புண்ணாம்
பாறிடு சந்து சந்தில்
பண்புறு நோவதாகும்
நேரிலங் கயரும் காமியம்
நிச்சய நோதல் செய்யும்
பாரினிலபான வாயு
பண்புறச் சேருமன்றே”

“சுக்கிலந் தனைய டக்கின்
சுரமுட னீர்க்கட்டாகும்
பக்கமா கைகால் சந்து
பாரநோய் வழியிறங்கும்
மிக்கமார் நோயுண்டாகும்
மிகுந்திடும் பிரமேகந்தான்
தக்கதோர் போதுமாகின்
தரித்திடும்வாயுக் கூறே”.

SIDDHA MARUTHUVANGA SURUKKAM
PAGE NO.212

“அறைகிறேன் விந்தழிந்தால் மேகமாச்சு
அமுது வழிந்து சூடுகொண்டால் வாயு சேரும்
பறைகிறேன் சூலைகுட்டம் கிரந்திப் புற்று
பவுத்திர கள்பிளவை போட்டு கண்டமாலை
அறைகிறேன் அரையாப்பு ஒட்டிய புஜ
அருங்கரப்பான் சிரங்கு குன்மம் மாநீர்க்கட்டு
குறையவே நீர்கொண்ட அந்நோய் காசம்
குடிலமாம் பேதியோடு கிராணி பாண்டே”.

- Agathiyar vaithiya vallathy 600.
- PAGE No.175

The above poem describes that, when the semen is destroyed by body heat. Vatham will add with that, and many diseases will come, including Kalladaippu.

POTHU KURI KUNANGAL (General Signs & Symptoms)

Gradual or sudden obstruction to flow of urine, Unbearable pain (agonizing pain) in the penis, Excruciating pain and swelling is experienced at tip of penis if the calculus attempts to expel, Colicky pain radiating from loin to groin, lower abdomen urethra and genitalia, if the calculus is irregular with sharp projection it produce burning and scanty micturation with haematuria.

SYNDROMES ASSOCIATED WITH KALLADAIPPU

உக்காரச்சூலை

"குத்துமுக் காரசூலையின் குணந்தான்
கோர்வையாய் விலாவதனில் முதுகில் நெஞ்சில்
அத்தியினில் நாபியில பானமாங் குதத்தில்
அதிகத்துன் மாங்கிசந்தான் வளர்ந்து மேவிப்
பத்துமணந் படுக்கைபோற் சலத்து வாரப்
பதிநெருங்கி மூத்திரமாங் கிரிச்சி யுண்டாய்த்
தச்சுசடங் கடுப்பெடுத்து மதிக லங்கித்
தளர்ச்சியொடு மயக்கமாகத் தள்ளுந்தானே".

- SIDDHA MARUTHUVAM
- PAGE NO.325

Excessive growth of muscles in chest region, back of trunk, umbilicus and anal urethral orifice followed by stricture of urethral orifice like a sand like crystals blocked in urethra. Dysuria, body pain, impairment of conscious, tiredness and giddiness occurs.

5. CLASSIFICATION

In Siddha system, various types of kalladaippu are mentioned in various text books of Siddhars.

தோன்றிடதோர் நாலினிட நாமங்கேளாய்
சுறுக்கான வாதத்தின் கல்லடைப்பு
பூன்றியதோர் பித்தத்தின் கல்லடைப்பு
புரண்டதோர் சிலேட்டுமத்தின் கல்லடைப்பு
தன்னியதோர் சொந்தமா கல்லடைப்பு
தேகத்தைப் பற்றியே சிறிது காலம்
தான்றியே சலப்பையில் வந்தழிந்து
கருவியே லிங்கத்திற்றரிக்குந் தானே.

Yugi vaithya chindamani
PAGE NO.248

As per Yugi vaithya chindamani

1. Vali Kalladaippu
2. Azhal Kalldaippu
3. Iya kalladaippu
4. Mukkutra kalladaippu

SIGNS AND SYMPTOMS OF ABOVE CLASSIFICATION ARE

1.Vali Kalladaippu

“தரித்து நாபிக்குங் கீழ்ச்சுரக்காய் குத்திச்
சல மலந்தான் வீழாமற்றம்பமாகி
வரித்தும லிங்கத்தில் வலியுமாகி
மருவியதோர் பொத்தியெல்லா சுரந்துகட்டி
திரித்தியே கிடக்கொடாய் புரட்டலாகித
தேம்பியே மூச்சுமாய் வயிறு முப்பும்
உரித்ததோர் சதைபோல் உவர்ப்புமாகும்
ஓங்குகியதோ வாதக் கல்லடைப்புதானே”.

Yugi viathya chindamani 729
PAGE NO.284

Acute pricking pain in the lower abdomen, scanty urination, obstruction to the flow of urine, pain in the penis making the patient unable to sit. Patient will cry, swelling in the abdomen, Albuminuria will be present with mucous discharge and black coloured stone will be expelled.

2.Azhal Kalladaippu

“அடைப்பாகிச் சலந்தானு மருவலாகி
அயங்காய்ச்சிச் சொருகினாற் போலேகானும்
புடைப்பாகப் பொத்தியெங்கும் புழுக்கமாகிப்
பூட்டுப்போல் விகுவாகிப் பிரட்டலாகும்

மடைப்பாகி உதிரநிறமாய்க் கல்லாகி
வந்திழந்து லிங்கத்தில் மாட்டிக் கொள்ளும்
குடைப்பாகி குற்றலாய்க் கூச்சலாகிக்
குதட்டுமே பித்தக் கல்லடைப்புத்தானே".

Yugi vaithya chindamani 73
PAGE NO. 285

Obstruction to the flow of urine, burning sensation in the external meatus, acute pain in the urethra and excretion of small red coloured stones.

3. Iyya Kalladiappu

"தானான தொப்புளிலே வில்லுப் போலச்
சலியமாற் சுரந்துமே சற்றே குத்தும்
ஏனான காலொடு கைகள் சந்து
இடுப்பு தான் குடைச்சலா யிசிவு காணும்
வேனான லிங்கத்தின் வேண்மை தன்னின்
விறுவிறென்றே கடுப்பாகி வியர்வையாகும்
தேனான வெறுப்பக்கல் சிறுகல்லாகச்
சிக்கலாய் வந்திறங்கு சிலேட்டுமந்தானே".

Yugi vaithya chindamani 731
PAGE NO. 285

In this type of kalladaippu the symptoms are severe pain in the umbilicus, pain radiating towards thigh, pain in the joints, burning micturition, excessive sweating, small white coloured stones will come along with the urine.

4.Mukkutra Kalladaippu

"வந்திறங்கும் நீர்த்தாரையடி யிற்றானும்
மாவருத்த முண்டாகி வலியுமாகி
நொந்திறங்கி நீர்தானு மருவிப்பாயும்
நொய்தான சிறுமணற் போல் நொறுங்கிக்கல்லாம்

சந்திறங்கி நீர் வழியில் வந்து விழும்
தாக்கான சிறங்டகைக்கல் தினமொன்றுக்கு
தொந்தமாங் கல்லடைப்புச் சூட்டிட்டாயே".

Yugi vaithya chindamani
PAGE NO.286

Severe pain in Urethra, Dysuria, Oliguria, Crystals excreted in urine in the form of small sands, Handful crystals excreted in urine. It is a fatal disease.

The other name of Mukkutra kalladaippu is Venneer kalladaippu or Manar kalladaippu. It is mentioned in Aruvai Maruthuvam.

In Siddhar Aruvai Maruthuvam :
PAGE NO.112

1. Vali kalladaippu
2. Azhal kalladaippu
3. Iya kalladaippu
4. Venneer or Manar Kalladaippu

Classification According to Noi Vilakkam

"வளிமுதல் மூன்றினு தோன்றலாலும்
கருநீர் தன்னிற் தோன்றலாலும்
கல்லடை நால்வகைப் படுமென மொழியே"

There are 4 types of Kalladaippu according to Noi vilakkam

1. Vali Kalladaippu
2. Analaka kalladaippu
3. Iyya Kalladaippu
4. Karuneer Kalladaippu

வளிக்கல்லடைக் குறிகள்

“படர்மிகப் படுதல் பற்கள் கடித்தல்
நடுங்கல் உந்தியுங் குறியும் பிசைதல்
கசடுகீழ் வளியொடு கழலல் அழுதல்
சிறுநீர் துளித்தல் என்பவும் பிறவும்

வளியின் கல்லடைக் குறியென மொழிப.
(படர் நோவு துன்பம் - மலம் வளி – வாதம்)
கறுத்தஞ் சிவந்தும் முளைகள் பரந்தும்
வளியின் கல்லது வடிவுறு மென்ப".

Tongue Biting Palpitation and Shivering, Crushing pain of the lower abdomen and genital organ, drippling of urine, the stones are blackish red in colour.

அனலக் கல்லடைக்குறிகள்

“சுட்டென நீரியம் மிகவெம் பிடுதலும்
நோதலும் அனலைக் கல்லடைக் குறியே
சிவந்துங் கறுத்து மஞ்ச ளாகியும்
சேங்குரு வடிவில் கல்லது தோன்றும்
(தோன்றும் என்பதைக் தனித்தனிக் கூட்டுக)”

Burning Micturation, Dysuria, The stones are reddish black or yellow in colour and small in size.

ஐயக்கல்லடைக்குறிகள்

“நீரியங் குத்தல் திணித்தல் குளிர்தல்
எனுமிவை ஐயக் கல்லடைக்குறியே
வெளுத்தும் தேனிற மாகியு மொளிர்ந்தும்
பெருவடி வுடைத்தாம் ஐயக் கல்லடை”

Pricking pain, forceful pain with severe intensity when passing urine, Fever with rigor, White or honey coloured shining or luminant larger size stone expelled.

கருநீர்க்கல்லடைக்குறிகள்

“கருநீ ரட்ககலின் வளிசினந்தெழுந்து
விரைனளி னடுவில் அதுதனைத் தடுத்தலின்
கருநீர்க் கல்லடை மருவிடு மென்ப”.

Increased vatham, preventing ejection of semen.

MUKKUTRA VAERUPADUGAL :(Pathogenesis)

Disease occurs due to the derangement in

- Uyir thathukkal
- Udalthathukkal
- Kaalamaarupaadu (seasonal changes)
- Thinai(living lands) and
- Udal vanmai.

Mukkutra Iyal :

The function of the three uyir thathus:

a) Vali – (Kattru + Veli)
b) Azhal – (Thee)
c) Iyyam – (Neer+Mann)

The alteration of three thathu in their reaction to extrinsic or intrinsic factors results in disharmony. This altered harmony and balance variation of the three thathus results in disease. Their natural ratio (1 :½:¼) to each other is discerned by the physician at the wrist and each nadi is individually assessed for its strength, speed and regularity.

VATHAM

The term vatham denotes vayu, dryness, pain and flatulence. Based on functions and locations it is classified in to 10types. They are tabulated below.

S. No	Vatham	General Features	Changes in Kalladaippu
1.	Piranan	Responsible for respiration and it is necessary for proper digestion	Normal
2.	Abanan	Responsible for all downward forces such as voiding of urine, stools, semen, menstrual flow	Affected (scanty micturition)
3.	Viyanan (paravukaal)	Dwells in the skin and is concerned with the sense of touch... extension and flexion of the parts of the body and distribution, of the nutrients to various parts of the body	Normal
4.	Uthanan (melnokkukaal)	Responsible for all kinds of upward motion such as nausea, vomiting etc...	Affected (nausea, vomiting)
5.	Samanan (nadukkaal)	Considered essential for proper digestion, assimilation and carries the digested nutrients to each and every organ	Normal
6.	Nagan	Helps in opening &closing of eyelids	Normal
7.	Koorman	Responsible for vision, lacrimation and yawning	Normal
8.	Kirugaran	Induces appetite, salivation, all secretions in the body including nasal secretion and sneezing	Normal

S. No	Vatham	General Features	Changes in Kalladaippu
9.	Thevathathan	Induces and stimulates a person to become alert, get anger, to quarrel, to sleep etc	Normal
10.	Dhananjeyan	Resides in the cranium and produces bloating of the body after death. This leaves from the body after 3days of death, forming a way through the skull.	Normal

PITHAM

It is the thermal life force of the body. It is subdivided into five types. They are

S. No	Pitham	Normal Features	Changes In Kalladaippu
1.	Anarpitham	Peps up the appetite and aids in digestion.	Normal
2.	Ranjagapitham	Responsible for the colour and contents of blood.	Normal
3.	Saathagapitham	Controls the whole body and is held responsible for fulfilling a purpose.	Affected (dysuria,oliguria)
4.	Pirasagapitham	Dwells in the skin and concerned with the shine, glow, texture and its complexion	Normal
5.	Alosagapitham	Responsible for the perception of vision.	Normal

KABHAM

It is responsible for the stream lined functions of the body and maintains body's defence mechanism intact. It is again classified into 5 types.

S. No	Kabham	General Features	Changes In Kalladaippu
1.	Avalambagam	Lies in the respiratory organs, exercises authority over other khapas and controls the heart and circulatory system.	Normal
2.	Kilethagam	Found in stomach as its seat, moistens the food, softens and helps to be digested.	Normal
3.	Pothagam	Hold responsible for the sensory perception of teste.	Normal
4.	Tharpagam	Presents in the head and is responsible for the coolness of the eyes, sometimes may be referred to as cerebrospinal fluid	Normal
5.	Santhigam	Necessary for the lubrication and the free movements of joints.	Normal

PARUVAKALAM

S. No	Perum Pozhuthugal	Mukkuttra Marupaadugal
1.	Kaar kaalam (Aavani & purattasi) Aug 16 to Oct15	VATHAM-vettunilai vazharchi PITHAM-thanilai vazharchi
2.	Koothir kaalam (Iypasi &karthigai) Oct 16 to Dec15	VATHAM- thanilai vazharchi PITHAM- vettunilai vazharchi
3.	Munpani kaalam (Margazhi & Thai) Dec16 to Feb15	PITHAM- thanilai vazharchi

S. No	Perum Pozhuthugal	Mukkuttra Marupaadugal
4.	Pinpani kaalam (Masi& Panguni) Feb16 to June15	KABHAM- thanilai vazharchi
5.	Elavenir kaalam (chithirai & vaikaasi) April16 to June15	KABHAM- vettunilai vazharchi
6.	Mudhuvenir kaalam Aani & AadiJune16 to Aug15	VATHAM- thanilai vazharchi

THINAI (LAND)

Siddhars classified the lands in to five types. They are

1. Kurunchi - Mountain range
2. Mullai -Pastoral area of the forest
3. Marudham -The fertile river bed
4. Neidhal -The coastal region
5. Paalai - Arid desert

- The winter season gives good health to the man, early summer and latter rainy gives moderate health. Whereas early rainy and latter summer are more prone to diseases, that's why siddhars called it as Aanaga kalam Marudha nilam is the fertile area where no disease occurs

RELATION BETWEEN MUKKUTRAM, KAALANGAL AND THINAIGAL

Mukkutram	Paruvakalam (Seasons)			Thinai
	Thannilai vazharchi (Accumulation)	Vaetrunilai vazharchi (Aggravation)	Thannilai adaithal (Alleviation)	
VATHAM	Muthuvenil Kalam	Kaar kalam	Koothir kalam	Vatha disease is more prevalent in neidhal land
PITHAM	Kaarkalam	Koothir kalam	Munpani	Pitha disease is more prevalent in mullai land.
KAPAM	pinpani	Elavenil kalam	Mudhuvenil kalam	Kapha disease is more prevalent in kurinchi land

UDAL VANMAI (IMMUNITY):

Siddhars classify Udal vanmai as three types. They are

1. Iyarkai vanmai
2. Kala vanmai
3. Seyarkai vanmai

UDAL KATTUGAL

S. No	Udal Kattugal	General Features	Changes In Kalladaippu
1.	Saaram (digestive essence)	Responsible for the growth& development. It keeps the individual in good temperament and it enriches the bood.	Affected due to pain
2.	Senneer (blood)	Responsible for the colour of blood and for the intellect, nourishment, strength, vigour and valour of the body.	Normal
3.	Oon (muscle)	Gives lookable contour to the body as needed for the physical activity. It feed the fat next day and gives a sort of plumpness to the body	Normal
4.	Kozhuppu (fat)	Lubricates the organs to facilitate frictionless functions.	Normal
5.	Enbu (bones)	Supports & protects the vital organs, gives the definite structure of the body and responsible for the posture and movements of the body	Normal
6.	Moolai (bone marrow)	Nourishes the bone marrow and brain which is the centre that controls other systems of body	Normal
7.	Sukkilam/ Suronitham(sperm/ ova)	Responsible for reproduction	Normal

PINIYARI MURAIMAI (DIAGNOSIS)

Four steps are followed in diagnosing the disease. They are,

a. Poriyaal arithal

b. Pulanal therthal

c. Vinaathal

d. Envagaithervu

In detail,

a.Poriyaal arithal

In this the physician should carefully observe the changes that occur in the five sensory organs (Porigal) of the patient.

b.Pulanal therthal

The physician carefully applies his five senses of perception, smell, taste, vision, touch and sound to understand the condition of the patient.

c.Vinaadhal

The physician should interrogate about the patients name, age, occupation, socio economic status, food habits, history of past illness, history of present illness, family history, marital status, menstrual history and frequency of pain.

d.ENVAGAI THERVUGAL

நா நிறம் மொழி விழி மலமூத்திரம்

நாடி பரிசமிவை மருத்துவராயுதம்”

–நோய்நாடல் நோய் முதனாடல்–253

Nowadays advanced diagnostic tools have been developed by modern bio-medical scientists. But Siddhars have given eight diagnostic methodological tools. They are called as Envagai thervu.

Eight fold system of clinical assessments

Siddhars have given eight diagnostic methodological tools. They are,

1. Naa
2. Niram
3. Mozhi

4. Vizhi
5. Malam
6. Moothiram
7. Naadi
8. Parisam

GENERAL FINDINGS

1.NAA

Signs and symptoms in the tongue are noted here.

Color, salivary secretion, ulcers, coating, inflammation, taste changes, deviation and its nature are generally noted.

In *kalladaippu* the naa not affected.

2.NIRAM

The color of the skin is noted here.

In *kalladaippu* the niram may be affected in sukkila atchmari.

3. **MOZHI**

Character of the speech is noted, mainly uratha oli (high pitched), thazhntha olli(low pitched), or resembles the sound of any instrument.

In *kalladaippu* the mozhi will be affected to the patients who have severe pain leading to the thazhntha olli

4.**VIZHI**

Character of the eye is noted. Color, warm, burning sensation, irritation, visual Perception.

In *kalladaippu* the vizhi may be affected redness due to renal colic pain.

5.MALAM

The stools are examined for quantity; hardening (malakattu), loose motion (bethi), Color and smell.

In *kalladaippu* the malam will be affected due to either constipation or diarrhea.

6.**MOOTHIRAM**

a.Neerkuri

The urine is examined for its color, odour, volume, froth and weight.

In *kalladaippu* the moothiram is affected due to scanty micturation.

b.NEIKURI

அருந்து மாறி ரதமும் அவிரோதமதாய்
அக்கல் அலர்தல்அகாலவூன் தவிர்தழற்
குற்றளவருந்தி உறங்கி வைகறை
ஆடிக்கலசத் தாவியே காதுபெய்
தொருமுகூர்த்தக் கலைக்குட்படு நீரின்
நிறக்குறி நெய்குறி நிருமித்தல் கடனே"

–சித்த மருத்துவாங்கச் சுருக்கம் பக்கம்509

The early morning urine of the patient is analyzed by dropping a drop of gingely oil on the surface of the urine sample. The accumulation, formations, changes, and dispersal under the sunlight without any external disturbances of the urine sample can be noted.

- Vatha neer - The oil spreads like snake
- Pitha neer - The oil spreads like ring
- Kapha neer - The oil spreads like pearl
- If the oil spreads gradually, it indicates good prognosis
- If the oil spreads fast or gets mixed completely with urine or sinks in urine, it suggests bad prognosis.

Since *kalladaippu* is due to the derangement of vatham and pitham,the neikuri will be vatha or pitha neer.

7.NAADI

Naadi is responsible for the existence of life can be felt one inch below the wrist on the radial side by means of palpation with tips of index, middle and ring finger, corresponding to Vatham, Pitham, Kapam.

Three humors Vatham, Pitham, Kapam existing the ratio 1:½:¼ normally. Dearrangement in these ratio leads to various disease conditions.

Naadi nadai in Kalladaippu

"விழுகும் சிலநேரம் விடுபட்டு நீரோடும்
ஒழுகிய வாயுவும் ஒதுங்கினால் நோகாது
வழுகிய மந்தத்தால் வாயுவந்தே புகில்
கழுமி முதிர்ந்திடும் கல்லெரிப்பு ஆகுமே".

Thirumoolar karukadai vaidhyam
Page No.180

When the vatham add with mantham it produces the kalladaippu disease, Raththinachurakkam Naadi also describes aggravation of vatham produces the symptoms of Kalladaippu.

"ஏவலாய் குழுலாய் பித்தஞ் செய்குணம் விளம்பக்கேளாய்
கோலவேல் விழி சிவந்து குளிர்ந்திடிருக்கு மல்லால்
சீலவே நீர்கடுத்து நொந்து சுறுக்கெனச்சு வந்து விழும்
ஞாலமே கிறுகிறென்று நாவுலர்ந்திருக்கங் காணே".

Siddha maruthuva noi naadal noi mudhal naadal thirattu.
Page No.168.

It described aggravated pitham will produces the symptoms of Kalladaippu.

ஸ்பரிசம்: (Touch)

8.SPARISAM

By sparism the temperature of skin (thatpam – cold or veppam – heat) smoothness, roughness, sweating, dryness, hard patches, swelling, abnormal growth of organs and tenderness can be felt.

In Kalladaippu patients tenderness over the lower abdomen, renal angle and lumbar region.

Also patient's temperature is increased in lower abdomen sweating all over the body at the time of colic.

நீர்க்குறி (Urine examination)

Urinary examination is good diagnosis method compare to naadi and other Envagai thervugal. Thereiyar mention below as

நீர்க்குறிச்சிறப்பு

"தர்க்கசாத் திரிக ளானோர்
தங்களிற் றேர்ந்து நாடி
வர்க்கமாம் நாடி தன்னில்
வருவது மயக்க மென்றே
உற்றநீர்ப் பரிட்சை யாய்ந்தே
யுரைத்தன ரிதற்கு நேராய்
மற்றொரு விதிநு லில்லை
மருத்துவக் கலைவல்லோர்க்கே".

Theriyar Neekuri Neikuri Nool
Siddha maruthvanga surukkam
Page No.372

“அருந்துமாரிதமும் அவிரோதமாய்
அ.ஃகல் அலர்தல் அகாலவூண் தவிர்ந்தழற்
குற்றளவருந்தி உறங்கி வைகறை
ஆடிக்கலசத் தாவியேகாது பெய்
தொரு முகுர்த்தக் கலைக்குட்படு நீரின்
நிறக்குறி நெய்குறி நிருபித்தல் கடனே”.

Theriyar Neekuri Neikuri Nool
Siddha maruthvanga surukkam
Page No.334

Siruneer Should be collected in early morning ; patient should be eating six tastes of food with regular time and well sleeping over night, urine should be examine with in 3% hours.

Siruneerin pothugunam

“வந்த நீர்க்கரி எடை மணம் நுரை எஞ்சலென
றைந்தியலுளவை யறைகுது முறையே”

Theraiyar Neerkuri Neikuri Nool
Siddha maruthuvanga surukkam
Page No. 297

1. Niram (colour) 2.Eadai (Specific gravity) 3. Nurai (Froth) 4. Natram (Smell) 5.Enjal (Deposits)

Above the five parameters by which each urine sample should be examined.

NIRAM (COLOUR)

NIRA THOGAI

“பீதம் செம்மைபைங் கருமை வெண்மையென்
றோதைந் கொழுமையை யொத்துகு நீரே”

Theraiyar Neerkuri Neikuri Nool
Siddha maruthuvanga surukkam
Page No. 298

1.Yellow

2.Red

3.Green

4.Black

5.White

Urine may be any colour mention above

கல்லடைப்பு நீரின் குணம் (COLOUR INDICATING URINARY STONES)

The urine colour would look like flesh washing water this is indicated in kidney diseases.

"தீப்புலால் கழுநீர்ச் செயலெனிர் குண்டிக்
காய்த்துர்ப் பலத்தால் கதித்த நீராமத்
துர்ப்பலக் கபமும் சோரியும் கொதிப்புறப்
பற்பகலாகப் பையப் பதிந்தே".

Theraiyar Neerkuri Neikuri Nool
Siddha maruthuvanga surukkam
Page No. 341

EADAI (SPECIFIC GRAVITY)

Urine not thick is considered healthy.

"மிகத் தடிப்பும் மிகத் தேறலும் இன்றெனில்
சுகத்தைத் தரும் மெய்ச் சுபாவ நீர் நன்றெ".

Theraiyar Neerkuri Neikuri Nool
Siddha maruthuvanga surukkam
Page No. 344

NURAI (FROTH)

"பந்தமெய்ப் பசையிளகப்படும் பருவத்
தந்தர்ப் பூதமாய் அனில முத்திரத்தில்
சம்பந்தப்படும் ததிநுரைப் புனலே".

Theraiyar Neerkuri Neikuri Nool
Siddha maruthuvanga surukkam
Page No. 346

Urine may be frothy in nature, if it is reduced in vali, azhal and ayyam are said to be deranged.

NAATRAM (SMELL)

மணவிலக்கணம்

"ஓதமணத்தோ டவவோத மொத்தி றங்கும்
சீதளஞ் கம்மிய தேகிகளுக்கே
காணிதில சீழுற் கலந்திழி மணமுறின்
கருப்பநா பிகளுளுங் காமநா ளத்துளும்
விரணமுண் டின்றேல் எய்து மசுமரியல்
திருத்தலே திண்ண மெனமனத் துன்னே".

Theraiyar Neerkuri Neikuri Nool
Siddha maruthuvanga surukkam Page No. 345

Foul odour with pyuria is observed in patients with urinary lithiasis associated with urinary tract infection and ulcer.

ENJAL (DEPOSITS)

If urine excretion look like cured water white colour and sand like deposits in urine indicate stones in kidney. This mention as

"நார்த்தி நீர்ப்பால் போல
நனவுற்றங் கிழியு மானால்
மாரற்ப முற்ற நீரி
லடி மண்டிக் கிடந்த தானால்
பாரிந்த மெழுகு மாங்காய்
பற்றிய கல்வி னாலே
சீருற்ற செய்கை யென்று
தெரிவுறச் செப்ப லாமே".

- சித்த மருத்துவாய்கச் சுருக்கம்
Page No.575

NEI KURI

The urine kept on the kidney tray in sun light, on non wind condition, should be examined by dropping a drop of gingili oil gently with rod. If oil spread like snake, it indicates valineer, a ring indicates azhal neer, and float like a pearl it indicates iyya neer and sinks in urine indicates mukkutram.

"அரவென நீண்டின.்தே வாதம்
ஆழி போற் பரவின் அ.்தே பித்தம்
முத்தொத்து நிற்கின் மொழிவதென் கபமே".

- சித்த மருத்துவாங்கச் சுருக்கம்

Page No.532

In Kalladaippu patients, oil spreading like ring indicates Azhal neer or snake indicates Vali neer.

NOI KANIPPU VIVAADHAM

(Differential diagnosis of kalladaippu

1.நீரடைப்பு
2.நீர்க்கட்டு
3.நீர்ச்சுருக்கு

சாத்தியம், அசாத்தியம் (PROGNOSIS)

"சிட்டியசாத் தியத்தச் சொல்லக் கேளாய்
சூலுக்காகும் வாதத்தின் கல்லடைப்பு
பூட்டிட்ட பித்தத்தின் கல்லடைப்புப்
புகழானசேட்டுமத்தின் கல்லடைப்பு
முட்டிட்ட இதுமூன்றும் சாத்தியமாகி
முனையான மருந்துகளிற் செம்மை யாகும்
தோட்டிட்ட தொந்தமாங் கல்ல டைப்புத்
தொடுசுறுவே கொல்லுமிது சூட்சந் தானே".

Yugi Vaidhya Chintamani
Page No.225

According to Yugimunivar, vali, Azhal, and Ayya Kalladaippu are curable where as Mukkutra Kalladaippu is incurable.

மருத்துவம்; (LINE OF TREATMENT)

The entire siddha system of medicine consists of three great subdivisions namely,

1) Noyillaneri (preventive) – Kaappu
2) Noineekkuneri (curative methods) – Neekkam
3) Uramaakkumurai (strengthening methods) - Niraippu

Noyillaneri is the special approach of the siddha system where regular dietary habits , early rising , physical and mental discipilinaries are all emphasized. Prevention can mostly save our body and soul, but modernization results in alteration of good health, leads to disease,

Siddha system is playing major role in treating and preventing many chronic diseases. Like wise , Herbal medicines have several phyto chemicals which exert their beneficial effect on urolithiasis by multiple mechanisms like,

- Diuretic activity
- Crystallisation inhibiting activity
- Lithotriptic activity
- Antimicrobial activity
- Analgesic and anti inflammatory activity
- Improving renal function
- Regulates oxalate , Calcium mechanisms.

The main object of treatment is to bring down the deranged mukkutrams to natural equilibrium by giving purgatives, which cure derangement of vatham, this one of the cause for Kalladaippu.

As per the above mention, author gives purgation to all patients as their body condition, the author of dissertation has selected trial drug Agasthiar kulambu 50mg with Rice water empty stomach oneday only.

In Siddha sytem treatment is not only removable of disease but also the prevention and improving the body condition after removal of disease. This is said as kappu, neekkam and niraippu.

Fomentation

An attack of renal colic may be aborted by the application of heat fomentation (hot water bottle or heater) to the lumber region Immediate treatment of loin pain or renal colic is bed rest.

Prevention

1. For prophylactic purpose it is necessary to eliminate all hindrances to a free drainage of urine (constricition, adenoma of the prostrate etc) and to remove foci of infection from the teeth and tonsils.

2. To prevent the formation of urate calculi a diet of milk and vegetables and mineral water is prescribed.

3. In the presence of oxalate calculi restrictions are imposed on foods rich in calcium (Milk, raw eggs, potatoes) with total abstinence from chocolate, spinach gooseberries and carrots.

4. A patient with phosphorus, carbonate stones is kept on a meat diet and much water to drink.

Advice

1.Patients should drink large amount of water (4 lit / day)

2.Patient should not suppress the excretion of urine and seminal fluid.

3. Preparation containing Vit. D.Must be avoided.

4. Regarding prevention Anubhava vaidhya deva ragasiyam states that one should not suppress the excretion of Moothiram (Urine) and Sukkilam (Seminal fluid)

DIET FOR KALLADAIPPU PATIENTS

Do's

1. Drink 2-3 times of water daily.

2. Drink lemon juice, grape, sugarcane, cucumber juice with seeds melon juice, tendercoconut, barely water, plaintain pith juices.

3. Eat the following vegetables and Greens.

    1. Raddish
    2. Ladysfinger
    3. Bottle guard
    4. Onion
    5. Sirukeerai
    6. Pasalai keerai
    7. Keeraithandu
    8. Coriander leaves
    9. Mint leaves

Dont's

1. Avoid drinking fluoride containing water
2. Aviod Milk & its products, Fish and other seafoods.
3. Aviod cabbage, cauliflower, tomato seeds, mushroom.

MODERN ASPECTS

ANATOMY AND PHYSIOLOGY OF URINARY SYSTEM

KIDNEY

Kidneys are a pair of excretory organs situated on posterior abdominal wall, One on each side of the vertebral column behind the peritoneum. These organs are responsible for removing excess water, salt and waste products from the blood for maintaining its PH.

Location

The kidney is occupying the epigastric, hypochondrium, lumbar and umbilical regions. Vertically they extends from the upper border of T12 Vertebra to the centre of the body of L3 vertebra.

The Right kidney is slightly lower than the left kidney. And the left kidney is little nearer to the median plane than the right. The transpyloric plane through the upper part of the Hilum of Right kidney and the lower part of the hilum of Left kidney.

Size, Shape, Measurement

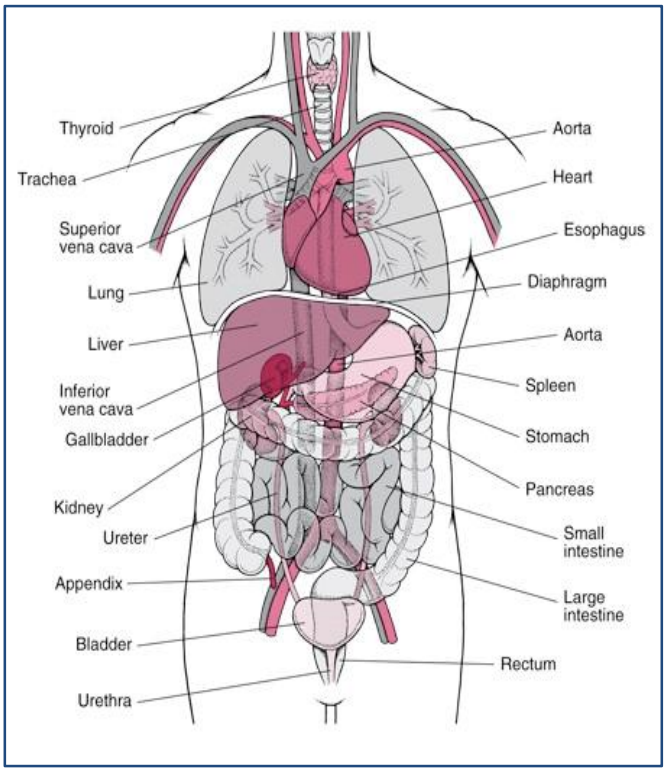
Each kidney is Bean shaped it is 11cm length 6 cm breadth, 3 cm thickness. Weight of the kidney is 150gm in males, 135 gm in females.

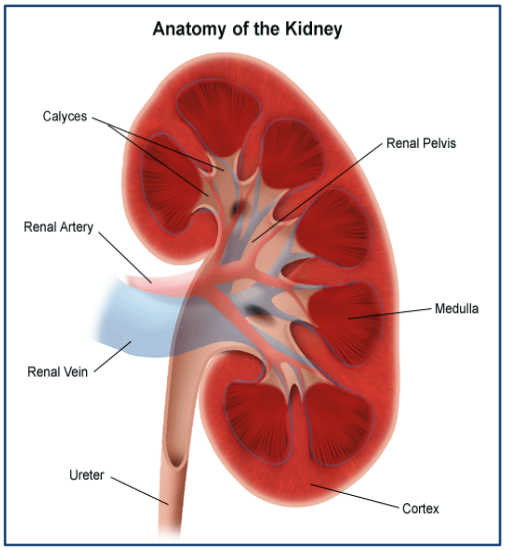
The Left kidney in little longer and narrower than the Right kidney because the long axis of kidney is directed downwards and laterally. So that the upper poles are nearer to the medium plan.

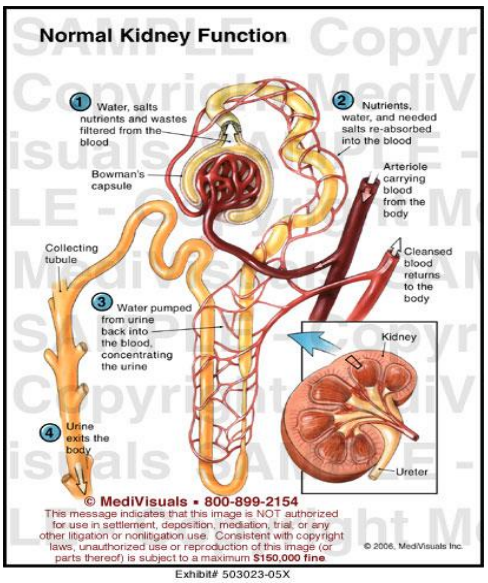
External features

Kidney are reddish brown in colour. Each kidneys has following features. It has 2 poles (upper and lower), 2 borders (medial and lateral), 2 surfaces (anterior and posterior).

ANATOMY AND PHYSIOLOGY OF URINARY SYSTEM







The upper poles are broad because they are compressed by corresponding supra renal glands, Lower poles are narrow

Lateral borders are convex and medical borders are concave with hilum in the middle. Anterior surface is some what irregular and posterior surface is flat.

Capsules of kidney

1. Fibrous capsule: This is a thin membrane which closely invests the kidney and lines the renal sinus.

2. Perirenal fat: This is layer of adipose tissue lying outside the fibrous capsule. It is thickest at the borders of the kidney and fills up the extra space in the renal sinus.

3. Renal fascia: This is a fibro aerolar sheath which surrounds the kidney and periorenal fat.

4. Pararenal body: It consists of variable amount of fat lying outside the renal fascia. It fill up the para vertebral gutter and forms a cushion for the kidney.

The depressed part present in medial border is called the hilum. It is about 2 to 2.5cm long. Hilum leads in to the renal sinus within the kidney. It is traversed by renal Artery, tributaries of renal vein and the Renal pelvis.

Renal pelvis is divided into 3 to 4 major calyces. These are divided into 7 to 14 minor calyces. Each minor calyces. Each minor calyx ends into an expansion, Which is intended by 1 to 3 renal papillae and is perforated by collecting tubules open on the summit of the Renal papillae. The Renal papillae are Nipple like projection in the wall of the renal sinus and represents the apices of the Renal pyramids.

Naked eye examination of the coronal section of the kidneys shows on outer reddish brown cortex and the inner pale medulla. The Renal medulla is made up of about 101-4 conical masses called renal pyramids. These apices forms the renal papillae which indend the minor calyces and discharge urine into them through the opening of the ducts of Bellini.

Histologically, each kidney is composed of 1-3 million uriniferous tubules. Each tubules are consists of two parts.

a. The secretary part which forms urine is called the nephron. The Nephron. The Nephron is the functional unit of the kidney.

b. Nephrons opens into collecting tubules.

The kidney cannot regenerate new Nephrons. Therefore with renal injury, disease or normal ageing, there is gradual decrease in nephron number. After age 40, the number of functioning nephrons usually decreases about 10 percent every 10 years. Thus at age 80 many people have 40 percent fewer functioning Nephorns than they did at age 40. This loss is not life threatening because adaptive changes in the remaining nephrons allow them to excrete the proper amounts of water, electrolytes and waste products.

Each Nephron contains, a tuft of glomerular capillaries called the glomerulus, through which large amount of fluid are filtered from the blood. A long tubule in which the filtered fluid is converted into urine on its way to the pelvis of the kidney.

The glomerulus contains a network of branching and anastamosing glomerular capillaries that, compared with other capillaries, have high hydrostatic pressure (about 60mm Hg). The glomerular capillaries are covered by epithelial cells, The total glomerulus's is encased in Bowman's capsule. Fluid filtered from the glomerulus's capillaries flows into Bowman's capsule and then into the proximal tubule which lies in the cortex of the kidney.

From the proximal tubule, fluid flows into the loop of henle, which dips into the renal medulla. Each loop consists of the descending and an ascending limb. The walls of the descending limb and the lower end of the ascending limb are very thin and therefore are called the thin segment of the loop of Henle. After ascending limb of the loop has returned part way back to the cortex, its wall becomes thick like other portions of the tubular system and it is therefore referred to as the thick segment of the ascending limb.

At the end of the thick ascending limb is a short segment, which is actually a plaque in its wall, known as the macula densa. The macula densa plays an important role in controlling nephron function. Beyond the macula densa, fluid enters the distal tubule that, like the proximal tubule lies in the renal cortex. This is followed by the connecting tubule and the cortical collecting tubule duct. The initial parts of 8 to 10 cortical collecting ducts join to form a single larger collecting ducts that runs downwards into the medulla and becomes the modularly collecting duct. The collecting ducts merge to form progressively larger ducts that eventually empty into the renal pelvis through the tips of the renal papillae into the renal pelvis through the tips of the renal papillae. In each kidney, there are about 250 of the very large collecting ducts, each of which collects urine from about 4000 nephrons.

Blood Supply

Usually there is one renal artery on each side, arising from the Abdominal Aorta. Accessory renal arteries are present in 30% of individuals; they arise commonly from the aorts, run parallel to the renal artery, and enter the kidney either at the hilum at one of its poles.

At or near the hilum the renal artery divides into anterior and posterior divisions. Further branching of these divisions gives rise to segmental arteries each of which supplies one vascular segment. Five such segments are described these are Apical, Upper, Middle, lower and Posterior. The segments are independent units.

Venous Drainage

The capillaries surrounding the tubules from various plexus and there form veins which accompany the corresponding Arteries forming the interlobular, actuate and interlooper veins ultimately to form the renal vein near the hilum and emerge out from it and open into inferior Venacava.

Nerve supply

The kidney supplied by the renal plexus, and offshoot of the celiac plexus. It contains sympathetic (T10-L1) fibers which are chiefly vasomotor. The afferent nerves of the kidney belong to segments T10-T12.

Mechanism or Urine formation

The process involved in urine formation are,

1. Glomerular filtration
2. Tubular secretion
3. Tubular reabsorption

Glomerular filteration

Glomerular filterate is a protein free plasma. Glomerular filteration is depend upon hydrostatic pressure of the afferent arterioles, the plasma proteins causing Osmotic pressure renal tubular pressure. The glomerular filter contains all the substance present in the plasma except colloids.

About 170 liters of glomerular filterate enters the renal tubule per day and about 168.5 liters of urine reabsorbed in the renal tubule.

Normal amount of urine excreted per day is about 1.5 litres. The glomerular filterate is alkaline. It contains water, small quantities of urea, glucose, potassium, calcium, bicarbonates and uric acid.

Tubular reabsorption

99% of glomerular filterate is reabsorbed in the renal tubule. Active reabsorption of sodium, potassium, glucose, amino acids, phosphate, calcium and uric acid occurs in the proximal convoluted tubule. 7/8 water present in the glomerular filterate absorbed at proximal convoluted tubule. This is called obligatory fraction.

Passive reabsorption of 80% of water is diffused out of the tubule. Thus the fluid within the tubule takes with it urea to diffuse out of the tubule. Sodium chloride concentration becomes grater within loop of Henle. Then the sodium chloride is reabsorbed by blood vessels accompanying the loop of Henle (Vasa recta). Along with this water also enters the vasa recta group of blood vessels.

In the distal convoluted tubule reabsorption of 1/8 of water occurs in the influence of anti diuretic hormone secreted by the posterior lobe of pituitary gland.Bicarbonates and chlorides are also reabsorbed in the distal convoluted tubule.

Tubular secretion

This occurs commonly within the convoluted tubule. When any substance is found excess in the blood, it is secreted into the urine Drug, mercurial diuretics, ammonium, potassium, hydrogen ion etc. are excreted by tubular secretion.

FUNCTIONS OF KIDNEYS

Kidneys perform vital functions. By excreting urine, kidneys play principal role in the maintenance of Internal environment. In addition, kidneys perform many other functions as described below.

Role in Homeostasis

The primary function of kidneys is homeostasis. It is accomplished by the formation of urine. Kidneys are not only the excretory organs, but are also the regulatory organs their major role is in homeostasis. During the formation of

urine, kidneys regulate various activities in the body, which are concerned with homeostasis.

i.Excretion of waste products

Kidneys excrete the unwanted waste products which are formed during metabolic activities.

a. Urea - End product of amino acid metabolism

b. Uric Acid - End product of nucleic acid metabolism.

c. Creatinine - End product of metabolism in muscles.

d. Bilirubin - End product of hemoglobin degradation.

e. Products of metabolism of other substances.

Kidneys also excrete harmful foreign chemical substances like.

a. Toxins
b. Drugs
c. Heavy metals
d. Pesticides etc.

ii.Maintenance of water balance

Kidneys maintain the water balance in the body by conserving water when it is decreased and excreting water when it is excess in the body.

iii.Maintenance of Electrolyte Balance

Maintenance of electrolyte balance, especially sodium in relation to water balance. Kidneys retain sodium if the osmolarity of body water decreases and eliminate sodium when osmolarity increases.

iv.Maintenance of acid Base balance

The pH of the blood and body fluids should be maintained within narrow range for healthy living. Body is under constant threat to develop acidosis, because of production of lot of acids during metabolic activities. However it is prevented by kidneys, lungs and blood buffers which eliminate these acids. Among these organs, kidneys play major role in preventing acidosis. In fact, kidneys are the only organs, which are capable of eliminating certain metabolic acids like sulphuric and phosphoric acids.

2.Hemopoietic function

Kidneys stimulate the production of erythrocytes by secreting erythropoietin. Erythropoietin is the important stimulating factor for erythropoiesis. Kidneys also secrete another factor called thrombopoietin, which stimulates the production the thrombocytes.

3.Endocrine function

The hormones secreted by kidneys are,

i. Erythropoietin
ii. Thrombopoietin
iii. Renin
iv. 1,25 dihydroxy cholecalciferol
v. Prostaglandins.

4.Regulation of Blood pressure

Kidneys play an important role in the regulation of arterial blood pressure, Kidneys regulate arterial blood pressure by two ways.

i. By regulating the volume of extracellular fluid
ii. Through rennin – angiotensin mechanism.

THE URETERS

The ureters are a pair of narrow, thick walled tubes which convey urine from the kidney to the urinary bladder. It lies deep to the peritoneum closely applied to the posterior abdominal wall in the upper part and the lateral pelvic wall in the lower part. Each ureter is about 25cm long (10 inches) and it measures about 3mm diameter. Upper half lies in the abdomen, the lower half lies in the pelvis.

Ureter begins within renal sinus as a funnel shaped dilatation called renal pelvis. It descends along the median margin and partly behind. It gradually narrows till the lower end of the kidney than the ureter passes downwards and slightly medially on the psoas major muscle and enter the pelvis by crossing in front of the termination of common iliac artery.

In the true pelvis the ureter at first runs downwards and slightly back wards and laterally, following the anterior margin of the greater sciatic notch. Opposite the ischial spine it turns forwards and medially to reach the base of the urinary bladder.

The ureter enter the bladder wall obliquely to open into it at the lateral angle of its trigone.

Normal Constrictions

The ureter is slightly constricted at three places. The first, at pelvic urethral junction about 5.5 cm below the hilum of the kidney. Second at Brim of the pelvis where the ureter crosses in front of the common iliac artery. Third is just before it enters the bladder. The renal stones tend to get arrested at these places.

Blood Supply

Upper part receives branches from Renal Artery, middle part receives from Aorta, the gonadal and iliac vessels. The pelvic part is supplied by the vessel, the middle rectal or uterine vessels.

Nerve Supply

The ureter is supplied by sympathetic (T10-L1) and para sympathetic (S2-S4). They reach the ureter through the renal, aortic and hypogastric plexuses. All the nerves appear to be sensory in function.

URINARY BLADDER

It is a hollow muscular organ which served to collect urine and to discharge to it out periodically. It lies in the anterior part of pelvic cavity. In front of Rectum in males and in front of uterus in females.

The bladder varies in its size, shape and position, according to the amount of urine is contained and the age of the person, when empty it lies entirely within the pelvis. But as if filled it expands into the abdominal cavity reaching upto umbilicus and becomes the abdominus.

Empty constricted bladder resembles 4 sided pyramid. It has 4 angles -Apex, Neck, Two Lateral angle, 4 surfaces – Superior, Posterior, 2 infero lateral surfaces, 4 Borders – Anterior, Posterior, 2 lateral Borders.

Distended bladder ovoid in shape. Since its angle and borders were rounded. In the fetus and new born even the empty borders abdominal. At age “6” bladder comes down to definite position and pelvic organ. Normal capacity of urinary bladder is about 200-300 etc.

Internal sphincter of the bladder

The bladder wall is made up of longitudinal and circular layers of smooth muscles and they are called detrusor muscle. In the trigone in addition to detrusor muscle. There is trigonal muscle of bell. There is original muscle of bell. There is no definite circular muscle fibre at the neck of the bladder stop at the level of neck. Longitudinal fibres from the posterior wall diverge to pass around the urethra on both sides.

Blood Supply

Superior vesicle arteries and inferior vesicle arteries supplies to the bladder. In addition branches from obturator and inferior gluteal Artery are supplied to the bladder.

Nerve Supply

Symphathetic fibres arises from T11-L2 segment. Parasymphathetic fibres branches from S_2-S_4.

URETHRA

Urethra is a tubular passage extending from the neck of the bladder to the external urethral orifice.

The male urethra extends from the internal urethral orifice at the neck of urinary bladder to the external urethral orifice at the tip of the penis. It is about 20 cm long in flaccid state of the penis the long axis of urethra shows 2 curvature and is therefore "S" shaped. In the erect state it become "J" shaped.

It is divided into 3 parts

1. **Prostatic part** : Passes through prostate (3cm long)
2. **Membraneous part** : Surrounded by sphincter (2cm long)
3. **Spongy part (penile part)** : Passes through the bulb and carpus spongiosum (15cm long)

Sphincter of the Urethra

There are 2 spincters, in relation with urethra internal and external. The internal sphincter made up of smooth muscle firbre and situated at the neck of the bladder is supplied by sympathetic nerves from lower thoracic segments and upper lumber segments.

The externals sphincter made of light striated muscle fibfe surrounds the membrancous part of urethra it is supplied by perineal branch of the pudental erve (S_2 to S_4)

Blood Supply

Branches of internal pudental Artery

THE FEMALE URETHRA

The female urethra is only 4cm long and 6mm in diameter. Developmentally it correspondents to the upper part of the prostatic urethra of the male.

It begins at the internal urethral orifices roughly 5cm behind the middle of the pubic symphysis. It runs downwards and forwards embedded in the anterior wall of the vagina, traverses the urogenital diaphragm and ends at the lateral urethral orifices in the vestibule.

RENAL CALCULI (UROLITHIASIS)

Definition

A condition in which one or more stones are present in the pelvis or calyces of the kidney or in the ureter.

Causes incidence and risk factors

A Kidney stone results when the urine becomes too concentrated and substances in the crystallize to form stones. Stones maynot produce symptoms until they begin to move down the ureter, causing pain. The pain, is severe, located in the flank and often described as worst pain ever experienced.

Kidney stones are common. About 5% of women and 10% of men will have atleast one episode by age 70. Kidney stones affect about 2 out of every 100 people. Recurrence is common and the risk of recurrence is greater if two or more episodes of kidney stones occur. Kidney stones are common in premature infants.

Some types of stones tend to run in families. Some types may be associate with other conditions which as bowel disease, ileal by pass for obesity or renal tubule defects. A personal or family history of stones is asassociated with increased risk of stone formation. Other risk factors include tubular acidosis and resultant nephrocalcinosis.

Calcium stones are most common accounting for 75% to 95% of the stones. They are two or three times more common in men, usually appearing at age 320-30. Recurrence is likely. The calcium may combine with other substances such as oxalate (the most common substance) phosphate or carbonate to form the stone. Oxalate is present in certain foods. Disease of small intestine increase the tendency to form calcium oxalate stones.

Uric acid stones may form in persons with cystinuria. It is a hereditary disorders affecting both men and women.

Struvite stones are mainly found in women as a result of urinary tract infection. They can few very large and obstruct the kidney ureter or bladder.

Aetiology:

Risk factors that enhance the stone formation are

Metabolic state (influenced by patient's genetic background) Common metabolic conditions that predispose to the formation of urinary stones are :

Idiopathic hypercalciuria is present in approx 50% of the stone forming patients in the urinary stones. It is divided into absorptive (due to excess GI absorption of Calcium) Renal type (due to renal leak of calcium)

Hyper Calciurea (with or without gout) is present in approx 30% of stone formers. Increased Uric acid excretion can also contribute to the formation of calcium containing stones.

Hyperoxaluria of various causes is present in about 15%

Low urinary citrate excretion is present in about 50% and can contribute to stone formation in most states.

Conditions associated with hypercalciuria and Hyperoxaluria

1. High dietary intake of calcium (dairy product)
2. Hyperparathyrodism
3. Hyper Vitaminosis D
4. Cushing's Syndrome
5. Renal tubular acidosis
6. Idiopathic hypercalciuria – Excessive absorption of calcium from a gut reduced respiratory tract absorption of filtered calcium

Hyper Oxaluria

1. High dietary intake of the fruit and vegetables.
2. Increased absorption of oxalate from gut
    a) Heat disease
    b) Low calcium diet

Hormonal imbalance

Hyper parathyroidism

This occurs due to increased secretion of parathyroid hormone by hyper active and hyperplastic tumours of parathyroid. In this condition there is increased urinary excretion of calcium and phosphorus. So it will deposit easily in urinary tract from the stone.

Environmental risk factors

A low urine volume is clearly with an increased risk of stones. Though not well documented there does appear to be an increased risk for patients in hotter climate or working conditions, at least during an initial acelimatization period.

Dietary Excesses

In Vit-D poisoning intestinal absorption of calcium and phosphorus increased. So more amount of Ca and P will be excreted via kidney by urine. So it will easily deposit and form stone easily.

Vit. A deficiency

It may cause a widespread atrophy of mucous membrance of Genito-urinary tract. When the mucous membrane of the urinary tract gets keratinized, the desquamated cells from the nidus on which salts of calcium and phosphous from urine precipitate and form calculus. This is more applicable to bladder calculus.

Urine pH effect urinary stone formation

In general an alkaline urine pH favours precipitation of inorganic stones – calcium, phosphate, and magnesium ammonium phosphate (Struvite). An acid pH>5.5 favours precipitation of organic stones uric acid and cystine.

Urine pH has little effect on calcium oxalate solubility and therefore little influence on formation of these stones.

Chronic UTI

a) Infection disturbs the colloid content of the urine, so there is more chance of the stone formation.

b) Infection also causes abnormalities in the colloids, which may cause the crystalloid to be precipitated.

c) Infection also change urinary pH, which helps in stone formation (eg. Proteus and staphylococcus)

d) Increased in concentration of crystalloids, which may under some circumstances produce stones.

Altered urinary Crystalloid and colloids

In urine there are quite a number of different types of crystalloids. These are kept in solution by the patients of colloids in the urine by process of absorption.

Urinary crystalloids are (eg) oxalate, calcium, cystine, uric acid, phosphate etc.

Urinary colloids are – mucin, chondroitin, sulphuric acid.

When there is imbalance in the crystalloid – colloid ratio, either there is an increase in the crystalloid level or a fall in the colloid level: urinary stones may formed.

If there is any modification in crystalloids eg. If they loose their solvent action or adhesive property, urinary stones may develop.

Urinary Stasis

Stones are more prone to occur when there is obstruction to the free passage of urine.

1. Urinary staisis provide a fertile field for bacterial growth and also predispose to urinary infection.
2. It also shifts the pH of the urine into alkaline side.
3. It allows crystalloids to precipitate

Mechanical Cause

Three mechanism currently thought to contribute to urinary stone formation are

1.Precipitation - Crystallization from Super Saturated Solutions.

2.Absence of inhibits of stone formation normally presents in urine

3.Presence of macromolecular matrix

Precipitation of a substance to form stones depend on many factors including solubility, concentration and urine characteristics i.e. pH 7

Normal constituents of urine that inhibit stone formation include citrate pyrosphosphate and magnesium. Reduced concentration of these substances are felt to contribute stone formation. Disease caused Renal Calculi is Gout – it forms the urate Calculus.

Types of Stones

1. Primary Stones
2. Secondary Stones.

PRIMARY STONES

Which appear in apparently normal urinary tract without any antecedent inflammation. These stones are usually formed in acid urine. These stones usually consist of

1. Calcium oxalate
2. Uric acid and Urate Calculi
3. Cystine Calculi
4. Xanthine Calculi
5. Indigo Calculi

Uric acid and Urate Calculus

Uric and stones form in acidic urine. In Israle, the incidence is as high as 25% Dalmation coach dog is the only mammal, at risk for formation or uric acid urinary lithiasis.

The risk is roughly equal to that of human do not possess the hepatic enzyme uricase, found in other mammals which form the water insoluble uric acid into ailantom, which is freely soluble and excreated by the Kidney. The consequency of this enzyme defect is that humans and Dalmation dogs have uric acid levels that are 10 times greater than those of other mammal.

Etiology

Uric acid is the end product of purine metabolism and the supersaturation of urine with the undissociated uric acid is needed for the development of uric acid crystals.

Diet restriction

Red Meat

Beef

Chicken

Fish

Peanuts and

Liver which are rich in purine.

Cystine Calculi

Cystine stones are rare only 1-2% have cystine stones.

Causes

Cystine stones result from an inherited defect of renal tubular reabsorption of cystine. Actually the tubular defect affects the reabsorption of the following 4 dibasic amino acids.

1. Cystine
2. Ornithine
3. Lysine and
4. Arginine

In above these amino acids, only cystine is insoluble and will precipitate in urine and to form stones.

In an autosomal recessive fashion, if both parents are carriers the sibling may have a 25% chance of being affected.

Management

3-4 quarts of oral fluids per day should be ingested to decrease the urinary concentration of cystine.

The urine should be alkalinized because cystine is more soluble in alkaline urine.

Diet

Sulphur containing proteins such as meat, fish and Eggs should be restricted.

Carbohydrate and fats may be increased in the diet along with low sulphur content proteins.

Nature of Cystine Stone

Colourless

Dense consistency

Pure Cystine stone not radio opaque but with sulphur it will radio opaque.

Xanthine Calculi

Extremely rare

Smooth round and brick red colour

On cut surface it shows lamellar appearance

Xanthine is a precursor of uric acid. It is found in most bodily fluids. Xanthinuria is a rare hereditary disorder of purine metabolism due to deficiency of the enzyme xanthine oxidase which cause excessive urinary secretion of Xanthine will form Xanthine Crystals.

Indigo Stones

Indigo stones are uncommon that there are merely academic curiosities. This is blue in colour and are derived from Indicon, formed by decomposition of tryptophan in the intestine and found in the urine.

Tryptophan naturally occurring amino acid excisting in proteins and essential for human metabolism.

Now we can see the detailed facts about the calcium oxalate stones.

Calcium Oxalate Stones\

It is an inorganic stone, it forms in alkaline urine calcium oxalate calculi may first come to medical attention when they cause pain or Haemattururia. The symptoms usually result from acute obstruction of calcium. Stones may be found at radiography evaluation.

Nature of Calcium Stones

Calcium Oxalate

Colour strained black with blood pigments. It readily damage the mucous membrane.

Calcium phosphate

Greyish white colour, loose, crumbly rough. Very hard consistency.

Causes for formation of calcium Oxalate Stones

1. They form when the urine is super saturated with calcium oxalate i.e. when the concentration of calcium oxalate in the urine exceeds its solubility.
2. Hotter climates – A low urine volume is clearly associated with an increased risk of stone formation.

Diet restriction

1. A high fluid intake is most important as it will increase urinary volume and decrease the concentration of calcium oxalate.
2. Tea, nuts and some green leafy vegetables are high in oxalates.
3. Restriction of salt and fat intake.
4. Dairy products
5. Foods high in oxalates are Tomato, strawberries, plumbs, spinach etc.

FOODS AND DRINKS CONTAINING CALCIUM OXALATE

A diet low in oxalates are more reasonable than a calcium restricted diet.

Apples, Grapes, Asparagus, Ice Cream, Beer, Milk, Beets, Oranges, Berries, Peanut butter, Black pepper, Pine apples, Cheese, Spinach, Chocolate, Tea, Cocoa, Turnips, Coffee, Vitamin C, Cola drinks, Yogart, Figs. These foods can be eaten in very limited amounts under the advice of the doctor.

SECONDARY STONES

1.Phosphate calculus or struvite stones

A struvite stone is formed due to infection in the urinary tract and so it is a secondary calculus. Struvite stone is a mixture of magnesium ammonium phosphate and carbonate apatite. App. 5- 20%

Phosphate stone may occur as covering of a primary stone. Such stones are known as mixed stones. The primary stone is often the calcium oxalate stone. When the urine becomes infected deposits of phosphates occur on the rough surface of calcium oxalate stones.

Clinical Features

Symptom can be divided into 4 groups.

1.Quiescent Calculus

A few stones, particularly the phosphate stones, may ie. Dormant for quiet a long period. During the time the stones gradually increase in size with destruction of renal parenchyma. Such stones are may be discovered accidentally in X-ray performed for some other reason or first revealed with renal failure ureaemia.

Sometimes such stones are discovered due to symptoms of urinary infection. When the stone is still in the submucosal stage (randalls plaque) or adherent to the parenchyma, it may be symptomless. Even stag horn calculus may be asymptomatic.

2. Pain

It is a leading symptom of renal calculi in majority of cases (80%).

3 types of pain are usually noticed viz.

a) Fixed Renal pain
b) Ureteric Colic Pain
c) Referred pain

a) Fixed Renal pain

i) If the stone is free and obstructs a calyx or uretero pelvic junction this will be dull flank pain due to capsular and parenchymal distension.

ii) Dull aching or boring type of pain is also experienced in case of big phosphate calculus.

iii) The pain is situated in the renal angle posteriorly and in the corresponding hypochondrium anteriorly.

iv) This pain characteristically coarse on movement particularly walking up the stairs and during jolting.

b) Ureteric colic

1. It is due to the stone attempts to pass down the ureter and temporary blocks the pelvi ureteric junction.
2. It is an agonizing pain and radiates from loin to groin.
3. The pain comes on suddenly, during which the patients rolls and drawing up his knees towards the chest tossing on the bed in agony.

4. Profuse sweating, nausea and vomiting accompany this colic. The pulse quickens and temperature goes down below normal. The typical radiation of the colicky pain is due to reflex pain which takes place along the course, Of the iliohypogastric and ilioinguinal nerves which are the somatic nerves of the same segments which supply the autonomic nervous system to the ureter. (T_{11}, T_{12} and S_1)

5. Sometimes the pain is referred to the scrotum or labium majora and to the inner side of the thigh along the distribution of Genito femoral nerve when the stone is in the lower part of the ureter. In male the testes may be retracted by spasm of cremaster and tenderness may persist for some days after the colic ceased.

6. When the stone is in the intra mural part of the ureter strangury may occur. The colicky pain persists for a variable period usually 6-8 hrs and passes off as suddenly as it came. Ureteric colic may pass off with compensatory polyuria for passage of stones in the urine.

c) Referred pain

This is quite rare and is sometimes referred to all over the abdomen. Such pain may stimulate peptic ulcer or gall bladder disease. Some times pain may referred to the opposite kidney which is known as renorenal reflex.

3) Hydronephrosis

Sometimes partients complaints of a lump in the loin and a dull ache which are due to hydronephrosis caused by renal stone.

4) Haematuria

Is the leading symptom. It usually occurs in small amount to make the urine dirty or smoky during or after an attack of pain.

Infection of the kidney may occur due to stone, which is relatively symptomless. Patients presents with pus in the urine.

Physical Signs

In majority of cases characteristic physical signs are not present. The signs which may be present and should be looked for are

1. Tenderness

This is mostly present at the renal angle posteriorly. Tenderness is more a constant feature when renal calculus is associated with infection.

2. Muscle rigidity over the kidney may be found in a few cases. Rebound tenderness anteriorly can also be elicited, particularly if acute infection is associated with

3. Swelling – when there is hydronephrosis or pyelonephrosis associated with renal calculus a swelling may be felt in the flank.

URETERIC CALCULUS

A stone in the ureter always originates from the kidney. Ureteric colic is the main symptom of this condition. Haematuria may be complained of. Ureteric colic starts as soon as the stone enters the pelvic ureteric juctions and recurs at longer or shorter intervals so long as the stone remains in the ureter. Ureteric colic ceases when the stone is ejected into the bladder or the stone is impacted in the ureter. When the stone is in the upper 1/3 rd of ureter, pain starts in the loin or near the renal angle and gradually radiates to the groin. Pain is griping in nature and starts suddenly. The patients almost tosses over the bed in agony often associated with profuse sweating and nausea. Pain suddenly goes off almost as suddenly as it appearted. At a lower level, pain commences rather anteriorly just above the iliac crest and is referred along the two branches of the genitor femoral nerve to the testis in the male, and Labium majus in the

female and to the antero – medical aspect of the thigh in both sexes. The testis becomes retracted by the spasm of the cremaster. When the stone enters the intramural part of the ureter, pain is referred to the tip of the penis and the patient complains of strangury. When the stone becomes impacted, colic goes off, instead a dull ache araise according to the site of impaction. Such pain varies in intensity, increased by exercise and relieved by rest. In the right side this conditions may be confused with appendicitis.

On examination, tenderness and rigidity may be felt along the course of the ureter. A stone in the lower part of the ureter may be felt in rectal or vaginal examination.

Straight X-ray often reveals often stone alsong the course of ureter. Urography will reveal a non-opaque stone by filling defect. No excretion or delayed excretion in sometimes seen after an attack of ureteric colic. Cystoscopy will reveal a stone at the ureteric orifice. It will show delayed or no excretion after intra venous injection of indigocarmine.

VESICAL CALCULUS

Stone may come to the bladder through the ureter and enlarges here. Otherwise stone may form in the bladder secondary to stasis and infection.

No age is exempt from this disease. Males are much more often affected than females. Increased frequenc of micturation is the commonest symptom. This is not experienced at night. The cause is that in standing posture the stone comes in contest with the trigone an initiates desire to micturate. During night the stone falls off the trigone and frequent desire to micturate goes off. Presence of stone in the bladder will give rise to pain in the supra pelvic region particularly after micturation. This pain is often referred to the tip of the penis or to the Labia majora and becomes aggravated by running and jolting. Children may scream and pull the prepuce for after micturation. Haematuria at the end of the micturation to also common symptom. This is caused by

abrasion of vascular trigone and gets worse on exercise. Sudden interruption of the flow due to blockage of the urethral meatus with the stone and subsequent continuation by change of posture is also not uncommon. Symptoms of cystitis eg. Frequency of micturation, burning sensation, post public pain etc may overshadow. Those due to presence of stone.

Sometimes stone may be situated in the post – prostatic pouch or diverticulum without any typical symptom of stone an to only revealed in X ray or cystoscopy for other complaints. These are known "Latent Stone".

On examination, one may elicit suprapubic tenderness. A large stone can be felt per rectum (in male) or per vagina (in female). But bimanual palpation (one on the abdomen and other in the rectum may facilitate palpitation.

Urine should be examined microscopically and Bacterologically, Straight X ray will reveal about 95% of the vesical calculus further stone on the ureter or kidney. Urography will show non-opaque stone by filling detect and demonstrates the functioning conditions of the kidney. Cystocopy and bladder sound will detect presence of stone as well.

INVESTIGATIONS

1) Blood examination

Calcium, Phosphate, Uric acid, Urea and electrolytes, parathyroid hormone – only if calcium excretion is high.

2) Urine Analysis

Protein, Blood, Glucose, amino acids

a) Physical examination
b) Chemical examination
c) Microscopic examination
d) Bacteriological examination

3. Renal function tests – Blood Urea, Creatinine

4. Radiography :

KUB – cystine stones are not radio opaque. So it may not be detectable in X-ray.

Excretion urography

Intravenous pyelogram

5. Ultrasonography

6. Computed tomography

7. Renal scan

8. Ureteroscopy, cystoscopy – instrumental examination

9. 24 hour Urine

Urea, Cratinine Clearance, Sodium Calcium Oxalate. Uric acid.

10. Examination of stone.

11. MRU (Magnetic Resonance Urography)

12. Stone Analysis

Prevention and recurrence

A) False recurrence – which means a tiny stone may overlooked any where in urinary tract after treatment.

B) True recurrence

Preventive measures divided into 2 categories

1. General measures
2. Specific measures

General measures

Stone must be analysed chemically to know its composition. If not available, we should be carefully note the following.

1. X-ray density
2. Types of crystal found in urine
3. Chemical test for cystine crystal and
4. Abnormalities in blood chemistry (Excess of calcium phosphorus or uric acid)

In general measures or advises which should be given to the patient regardless of the type of stone are :

a. Fluid intake should be high at all times.
b. Urine should be kept acid all the time.
c. Vitamin –D should be stopped or used in very low quantity Specific measures (Diet restriction)

1) Calcium stone – Dairy products, fruits and vegetables rich in calcium.
2) Oxalate tones – Tomatoes, plums, spinach etc.
3) Uric acid and urate calculi - Red Meat, Fish, Liver

4. Cystine Calculus

Sulphur containing proteins such as meat, fish, egg should be restricted. Carbohydrate and fat may be increased with low sulphur content proteins.

For prophylactic purpose it is necessary to climate all hindrances to a free drainage of urine (constriction, adenoma of the prostrate etc) and to remove foci of infection from the teeth and tonsils.

DIFFERENTIAL DIAGNOSIS OF RENAL CALCULUS

1. Cystitis and urinary tract infection

2. Pyelonephritis

3. Perinephric abscess

Complications of Renal Calculus

1. Azotemiz : Increased urea and nitrogen containing compounds.
2. Acquired distal renal tubular acidosis
3. Urinary tract infection due to urinary stasis.
4. Hyperkalaemia
5. Renal hypertension
6. Chronic Unilateral or bilateral hydronephrosis in the presence of ECV expansion or other renal disease.
7. Polycythaemia – infrequent complication of obstructive uropathy secondary to increased erythropoietin production by the obstructed kidney.

Treatment of stone disease

The medical management of urolithiasis involves mainly two aspects : one that is aimed at dissolution of preexisting stones and there, at preventing the recurrence of stones. Surgical treatment is method of choice for removing previously existing stones while appropriate therapeutic treatment has to be chosen for prevention or recurring stones.

Surgical Treatment

Depending upon the location of the stone, either operative or endoscopic approach is chosen. Endoscopic method is preferred for stones located distal to the bladder. Some of the non – operative techniques devised are extra – corporeal shock wave lithotripsy (ESWL), precutaneous nephrolithotomy (PCNL) and Cystolithotripsy. These also cause side effects such as hemorrhage, hypertension, tubular necrosis and subsequent fibrosis of the kidney.

TRIAL MEDICINE

மேகராசாங்கசூரணம்

ஆதாரம்:ஆத்மரட்சாமிர்தம் ப.எண்:430

சேரும் சரக்குகள்:

வெட்டிவேர், சீரகம், சாதிக்காய், சாதிப்பத்திரி, சந்தனம், கர்கடகசிங்கி, தக்கோலம், நெல்லிவற்றல், சிறுநாகப்பூ, கோஷ்டம், குங்குமப்பூ, நிலப்பனைக்கிழங்கு, ***தாமரைமகரந்தம்,*** அமுக்கராக்கிழங்கு, பேரிச்சம்பழம், திராட்சைப்பழம், சுக்கு, மிளகு, திப்பிலி, வால்மிளகு, அகில்கட்டை, இலவங்கப்பட்டை, நெற்பொரி, ***பச்சைக்கற்பூரம்,*** கோரோசனை-இவை வகைக்கு½பலம் (17.5 கிராம்).

செய்முறை:

மேற்கண்ட சரக்குகளை இடித்துக்கலந்து வஸ்திரகாயம் செய்து எடுத்துக்கொண்டு அதற்குச் சமஅளவு சீனி கலந்துஎடுத்துக் கொள்ளவும்.

அளவு:2வேளை1 கிராம்

அனுமானம் :பசுவெண்ணெய்

நாள் அளவு: 1 மண்டலம் (48 நாட்கள்)

தீரும் நோய்கள்: கைகால் எரிவு, **கல்லடைப்பு**, நீர்த்தாரை வழி மோர்போல் இறங்கும் கற்பிரமியம் ,நீர்ச்சுருக்கு,உடல்வறட்சிஆகியநோய்கள் நீங்கும்.

Trial Medicine

(மேகராஜாங்க சூரணம்)

TRIAL DRUGS

ELAVANGAPATTAI

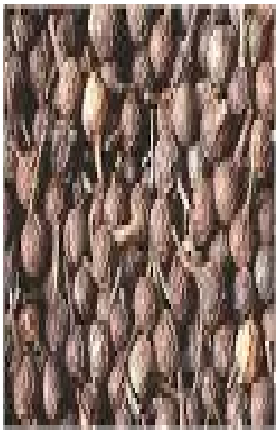
VETTIVER

SEERAGAM

SATHIKKAI

LAVANGAM

VAALMILAGU



PACCHAIKARPURAM

ADHIMATHURAM

KOROSANAI

JATHIPATTHIRI

KUNGUMAPPOO

SANTHANAM

AKHILKATTAI

SIRUNAGAPOO

THAMARAI

THIRATCHAI

AMUKKARA

BAERITCHAI

NILAPPANAI

KADUKKAI

NEL

NELLI VATTRAL

ACTIONS OF TRIAL DRUGS(According ToGunapadamMooligai)

Drugs	Botanical Name	Actions
வெட்டிவேர்	Veteveria zizanoides	சிறுநீர்பெருக்கி,இசிவகற்றி
சீரகம்	Cuminum cyminum	குளிர்ச்சியுண்டாக்கி
சாதிக்காய்	Myristica officinalis	உறக்கமுண்டாக்கிஅகட்டுவாய்வகற்றி
சாதிப்பத்திரி	Myristica officinalis	உறக்கமுண்டாக்கிஅகட்டுவாய்வகற்றி
சந்தனம்	Santalum album	சிறுநீர்பெருக்கிகுளிர்ச்சியுண்டாக்கி
சிறுநாகப்பூ	Mesua ferrea	வீக்கமகற்றி,துவர்ப்பி
கோஷ்டம்	Costus speciosus	சிறுநீர்பெருக்கி,உரமாக்கி
குங்குமப்பூ	Crocus sativus	இசிவகற்றி,துயரடக்கி
கிராம்பு	Syzygium aromaticum	இசிவகற்றி,அகட்டுவாய்வகற்றி
தாமரைமகரந்தம்	Nelumbo nucifera	உள்ளழலாற்றி,தாதுவெப்பகற்றி
கர்கடகசிங்கி	Rhussuceedenae	வீக்கமகற்றி,உரமாக்கி
தக்கோலம்	Takkolam	வெப்பகற்றி, துவர்ப்பி
நெல்லிவற்றல்	Phyllanthus emblica	சிறுநீர்பெருக்கி, குளிர்ச்சியுண்டாக்கி
நிலப்பனைக்கிழங்கு	Curculigo orchoides	சிறுநீர்பெருக்கி, துவர்ப்பி
அமுக்கராக்கிழங்கு	Withania somnifera	சிறுநீர்பெருக்கி, வீக்கமகற்றி
பேரிச்சம்பழம்	Phonex dactilifera	உள்ளழலாற்றி, குளிர்ச்சியுண்டாக்கி
திராட்சைபழம்	Vitis vinefera	குளிர்ச்சியுண்டாக்கி, சிறுநீர்பெருக்கி
சுக்கு	Zingifer officinalis	வீக்கமகற்றி,வெப்பகற்றி
மிளகு	Piper nigrum	வீக்கமகற்றி,நச்சரி
திப்பிலி	Piper longum	அகட்டுவாய்வகற்றிவெப்பமுண்டாக்கி
வால்மிளகு	Piper cubeba	சிறுநீர்பெருக்கி,அகட்டுவாய்வகற்றி
அதிமதுரம்	Glycyrhiziaglabra	உள்ளழலாற்றி,வறட்சியகற்றி
அகில்கட்டை	Aqullariaagallocha	வீக்கமகற்றி,உள்ளழலாற்றி
இலவங்கப்பட்டை	Cinnamomumzeylanica	உள்ளழலாற்றி, இசிவகற்றி
நெற்பொரி	Oryza sativa	உள்ளழலாற்றி,குளிர்ச்சியுண்டாக்கி
பச்சைக்கற்பூரம்	Borneo camphor	குளிர்ச்சியுண்டாக்கி,உரமாக்கி
கோரோசனை	Felbovinumpurifactum	குளிர்ச்சியுண்டாக்கி, இசிவகற்றி

CHEMICAL CONSTITUENTS OF THE MAIN DRUGS

(According to INDIAN MATERIA MEDICA)

VETTIVER – β- vetivone, α- vetivone.

(Diuretic , Antispasmodic)

NILAPANAI- Merketone,glycride

(Diuretic, emolient)

SANTHANAM- Santalol, β-sitostenol,

(Antimicrobial, Antiseptic, Antispasmodic)

NELLI- Tanin(Antidiabetic, Antioxidant)

VALMILAGU- (sesquiterpene, Hydro carbon diuretic)

KOSTAM- Diosgenine,β-Sitosterol,Saponins

(Diuretic, coolant ,Antihelmintic)

AMUKARA-Withanone, Withaferin.

(Antibiotic,Anti inflammatory)

MATERIALS AND METHODS

PROTOCOL

Study Designs

The open clinical trial on kalladaipu was carried out in the post graduate department of maruthuvam in govt. siddha medical college attached to arignaranna hospital of Indian medicine, Chennai-106. During the period of 2010-2012.

Sample Size

The study is conducted in 40 selected kalladaipu patients of both sexes between age groups of 20-60.

Selection Criteria

The patients having following parameters are selected for the study.

- Flank pain
- Burning micturation
- Nausea
- Vomiting
- Oliguria
- Dysuria
- Retention
- Haematuria
- Fever

Exclusion criteria

- Age group less than 15
- Staghorn calculus
- Calculi associated with elevated serum creatinine level
- Patient who does not complete the trial period
- Calculi in pregnancy

Proforma

The case sheet proforma for kalladaippu was prepared based on siddha methodology and modern aspect.

History taking

For better treatments & results a detailed clinical parameter was taken regarding history of present illness, past illness, family history, menstrual history, occupational history, socio economic status residential area, etc.,

Investigation

All patients were screened by the following investigations.
This was carried out regularly before and after treatment.

Urine examination

Albumin
Sugar
Deposits

Blood for Biochemical Examination

The blood was tested for sugar, urea, serum creatinine to know the renal function and its excretion.

Ultra sonogram

Ultra sonogram of complete abdomen including KUB was done in cases to know the location, size and number of calculi.

Drug and dose schedule

Megarajangachooranam- 1gm BD with butter for 48 days.

RESULTS AND OBSERVATION

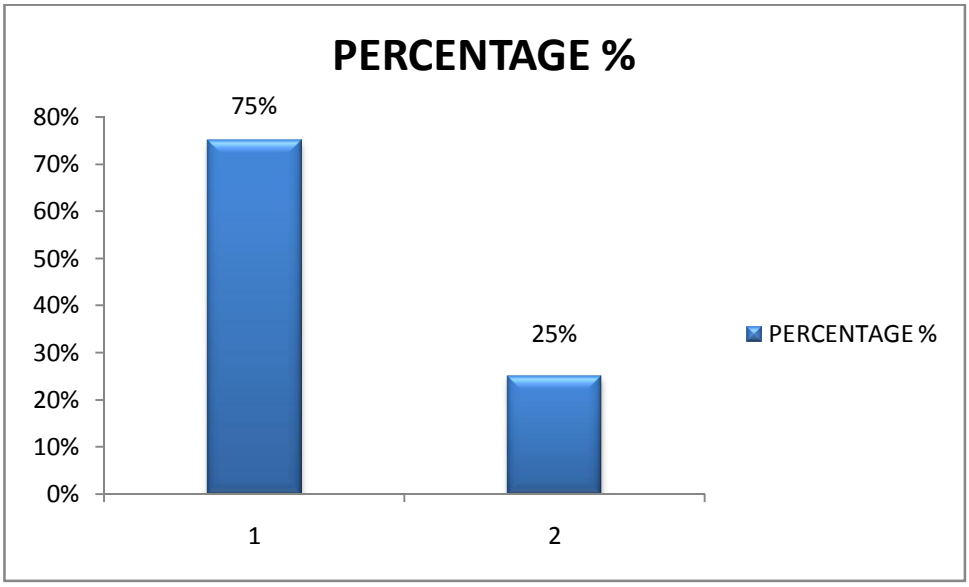
40 cases having kalladaipu at various places were selected and treated both OP and IP in PG maruthuvam department. Attached to A.A.G.H.I.M. Hospital, Chennai- 106 during the year 2010-2012. The result and observation during that clinical study are as follows.

1) Sex distribution
2) Age distribution
3) Occupation
4) According to season
5) Distribution of thinai
6) Distribution of mukkutram- vatham
7) Distribution of mukkutram- pitham
8) Distribution of mukkutram- kabam
9) Ezhuudalthathukal
10) Ennvagaithervugal- naadi
11) Ennvagaithervugal
12) Neikuri
13) Clinical manifestation
14) Distribution of calculi based on side
15) Distribution of calculi based on location
16) Results after treatment

1. SEX DISTRIBUTION

SEX	NUMBER OF CASES	PERCENTAGE %
Male	30	75%
Female	10	25%

SEX DISTRIBUTION



INFERENCE

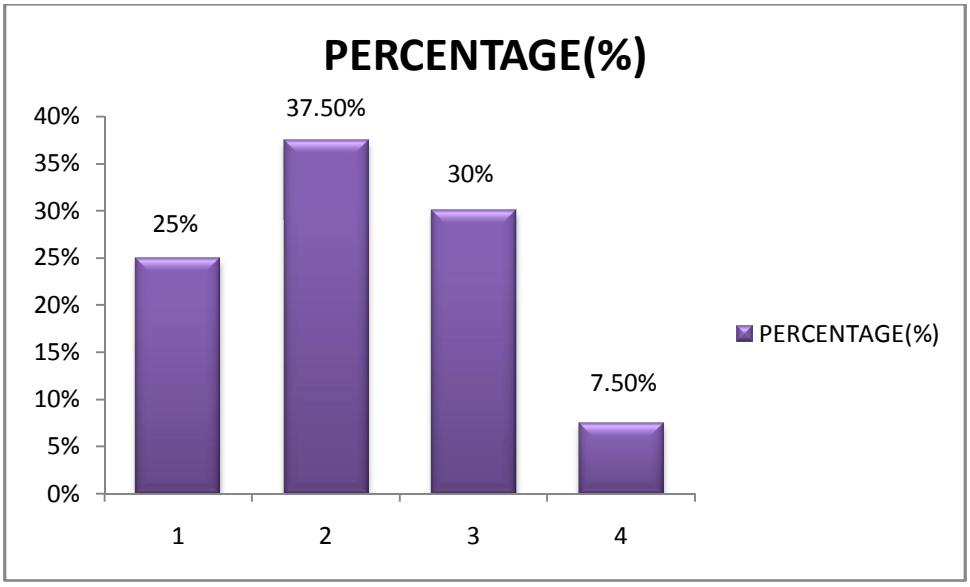
About 75% males and females were 25% .

Literature :According to Literature males more prone to renal calculi.

2. AGE DISTRIBUTION

AGE IN YEARS	NUMBER OF CASES	PERCENTAGE(%)
21-30 yrs	10	25%
31-40 yrs	15	37.5%
41-50 yrs	12	30%
51-60 yrs	3	7.5%

AGE DISTRIBUTION



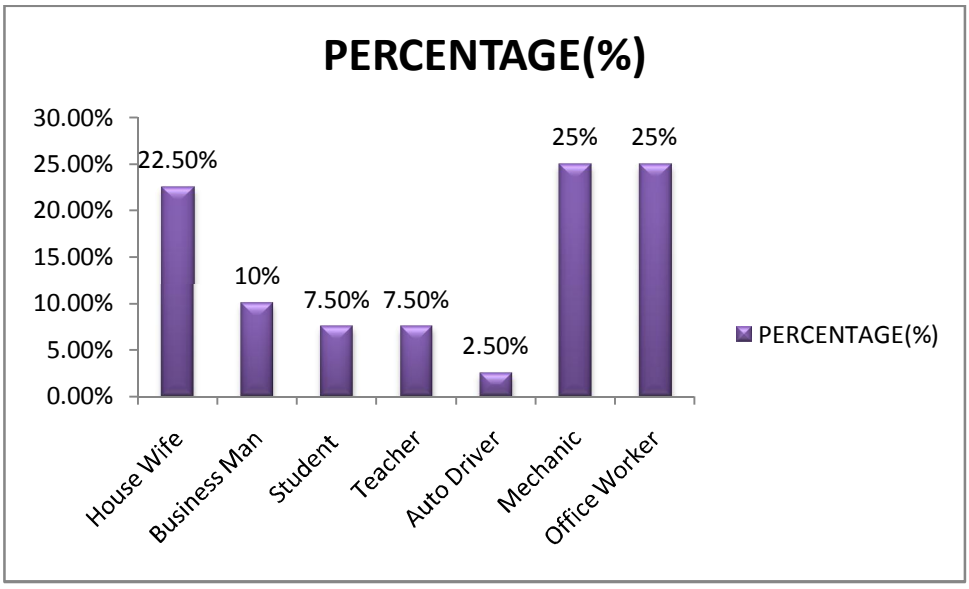
INFERENCE

Majority of the cases that is 37.5% were in the 3rd decade, 25% were in the 2nd decade, 7.5% were in the 5th decade and 30% were in the 4th decade.

3. OCCUPATION

OCCUPATION	NUMBER OF CASES	PERCENTAGE(%)
House Wife	9	22.5%
Business Man	4	10%
Student	3	7.5%
Teacher	3	7.5%
Auto Driver	1	2.5%
Mechanic	10	25%
Office Worker	10	25%

OCCUPATION



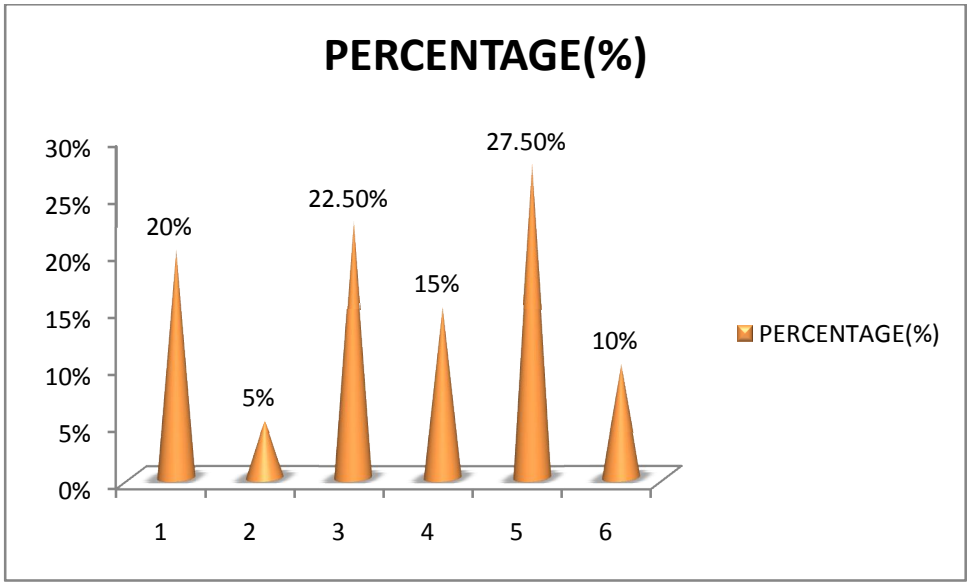
INFERENCE

25% Were Office Worker. 22.5% were Housewife.10% were Business Man.7.5% Were Students & Teachers.2.5% were Auto driver, 25% were mechanics.

4. ACCORDING TO SEASON

KALAM(season)	NUMBER OF CASES	PERCENTAGE(%)
Kaar Kaalam(Aug-Oct)	8	20%
Koothir Kaalam(Oct-Dec)	2	5%
Munpani Kaalam(Dec-Feb)	9	22.5%
Pinpani Kaalam(Feb-Apr)	6	15%
Elavenir Kaalam(Apr-Jun)	11	27.5%
Muthuvenir Kaalam(Jun-Aug)	4	10%

ACCORDING TO SEASON



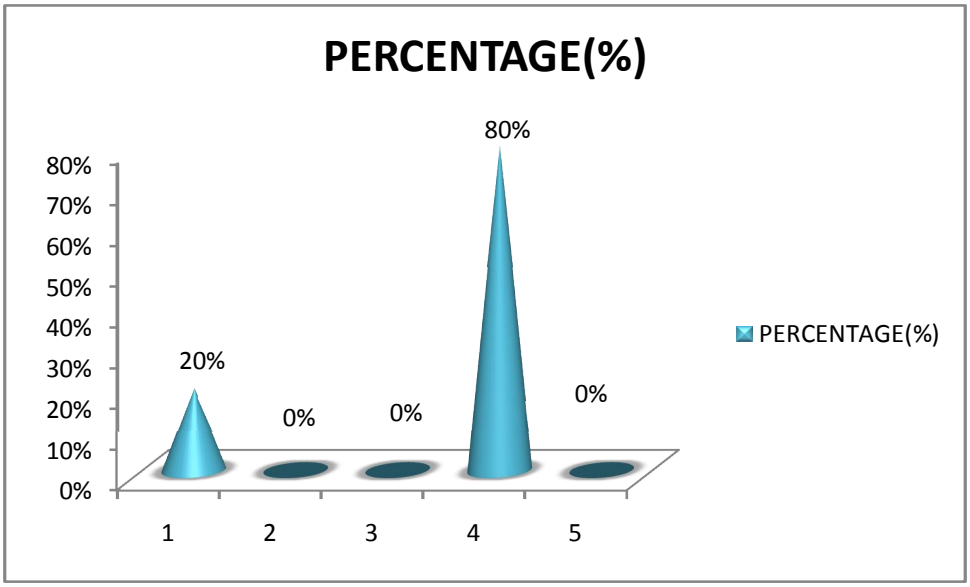
INFERENCE

According to paruvakalam highest incident of 27.5% were noted in elavenir kaalam and 22.5% cases were noted in munpani kaalam, 20% cases were noted in kaar kaalam.

5. DISTRIBUTION OF THINAI

THINAI	NUMBER OF CASES	PERCENTAGE (%)
Kurinchi	8	20%
Mullai	0	0%
Marutham	0	0%
Neithal	32	80%
Paalai	0	0%

DISTRIBUTION OF THINAI



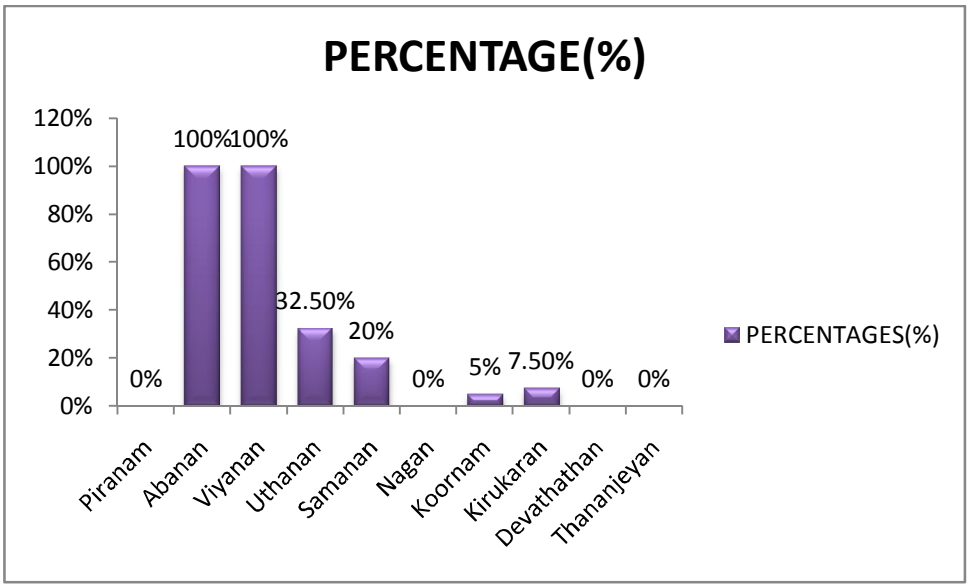
INFERENCE

According to thinai the highest distribution 80% was noted in neithal and 20% was noted in kurinchi.

6. DISTRIBUTION OF MUKKUTRAM- VATHAM

VATHAM	NUMBER OF CASES	PERCENTAGE (%)
Pranan	-	0%
Abanan	40	100%
Viyanan	40	100%
Uthanan	13	32.5%
Samanan	8	20%
Nagan	-	0%
Koorman	2	5%
Kirukaran	3	7.5%
Devathathan	-	0%
Thananjeyan	-	0%

DISTRIBUTION OF MUKKUTRAM- VATHAM

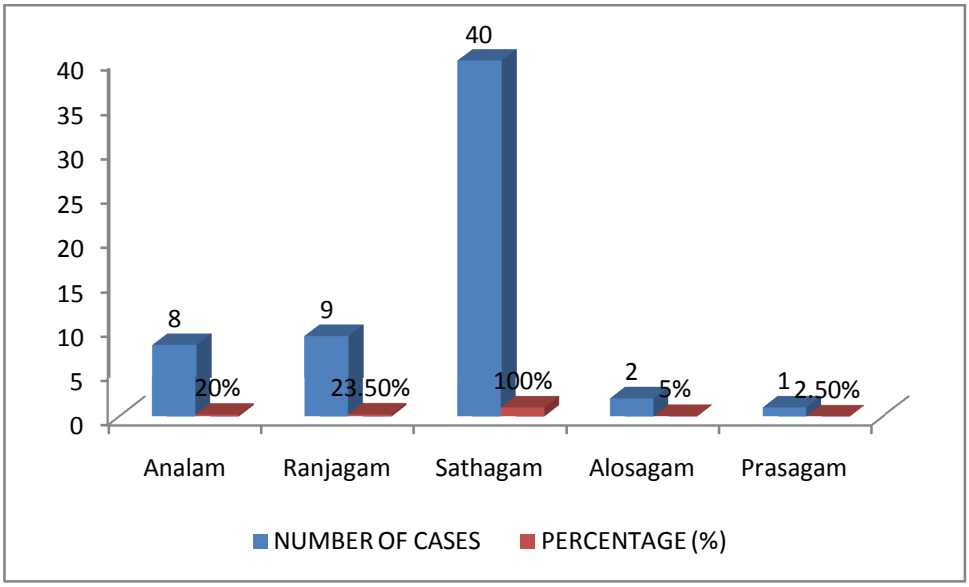


INFERENCE

According To Classification Of Vatham Derangement Of Abanan And Viyanan Were 100% And Uthanan Were 32.5%, Samanan Were 20%, Kirukaran Were 7.5%.

7. DISTRIBUTION OF MUKKUTRAM-PITHAM

PITHAM	NUMBER OF CASES	PERCENTAGE (%)
Analam	8	20%
Ranjagam	9	23.5%
Sathagam	40	100%
Alosagam	2	5%
Prasagam	1	2.5%



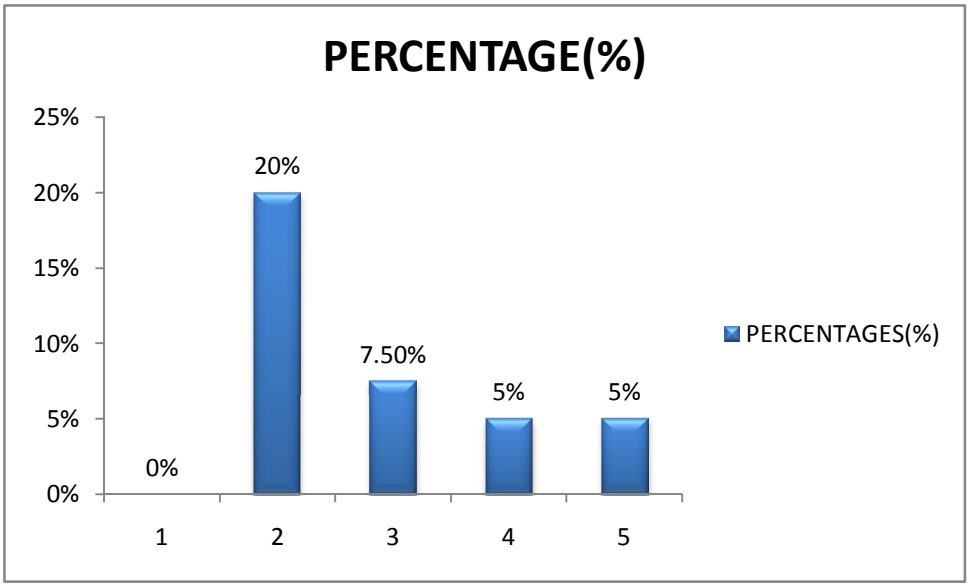
INFERENCE

According to pitham 100% of cases affected by the derangement of sathagam and 22.5% of cases were affected by ranjagam and 20% cases were affected by analagam (pallor &haematuria).

8. DISTRIBUTION OF MUKKUTRAM-KABAM

KABAM	NUMBER OF CASES	PERCENTAGE (%)
Avalambagam	-	0%
Klethagam	8	20%
Pothagam	3	7.5%
Tharpagam	2	5%
Santhigam	2	5%

DISTRIBUTION OF MUKKUTRAM-KABAM



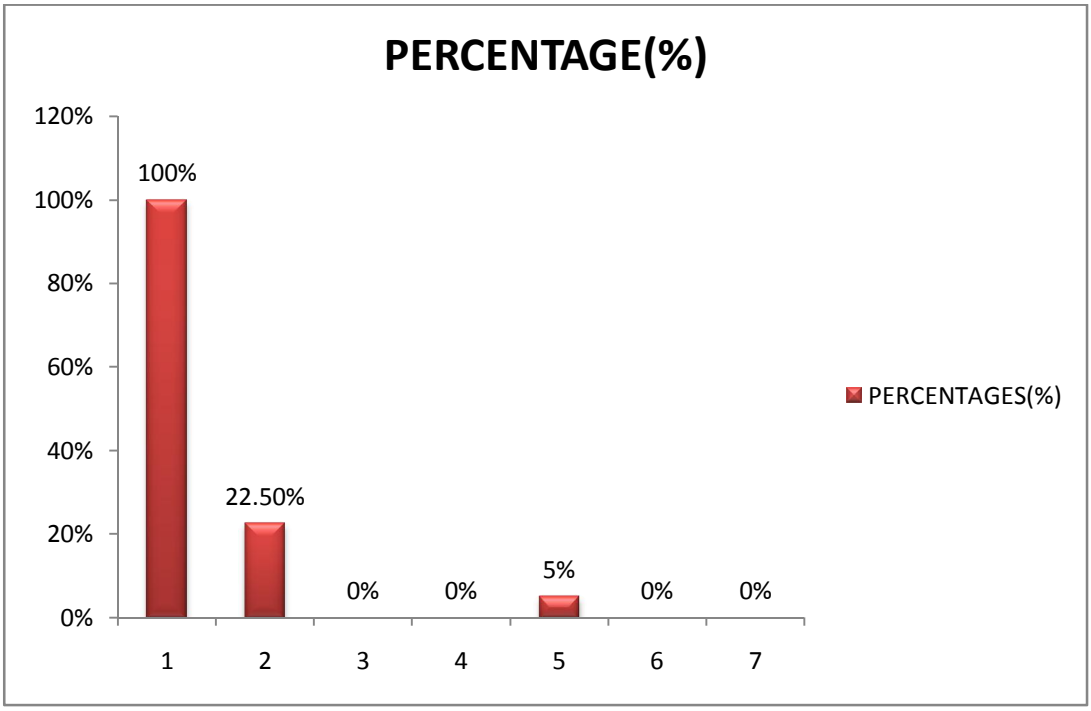
INFERENCE

According to kabam 20% klethagam was deranged 7.5% were affected by Pothagam and tharpagam and santhigam were 5%.

9. EZHU UDAL THATHUKAL

EZHU UDAL THATHUKAL	NUMBER OF CASES	PERCENTAGE (%)
Saaram	40	100%
Senneer	9	22.5%
Oon	-	0%
Kozhupu	-	0%
Enbu	2	5%
Moolai	-	0%
Sukkilam/Suronitham	-	0%

EZHU UDAL THATHUKAL



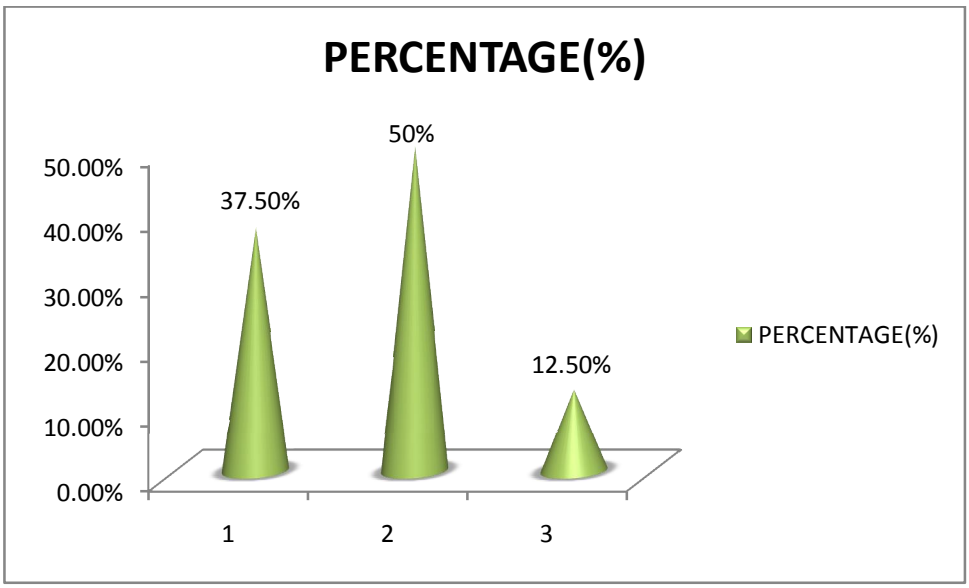
INFERENCE

Saaram Was Affected In 100% Of Cases. Senneer Was Affected In 22.5% Of Cases And Enbu In 5% Of Cases.

10. ENN VAGAI THERUVUGAL-NAADI

ENNVAGAI THERUVUGAL	NUMBER OF CASES	PERCENTAGE(%)
Vali azhal	15	37.5%
Azhal vali	20	50%
Iyya azhal	5	12.5%

ENN VAGAI THERUVUGAL-NAADI



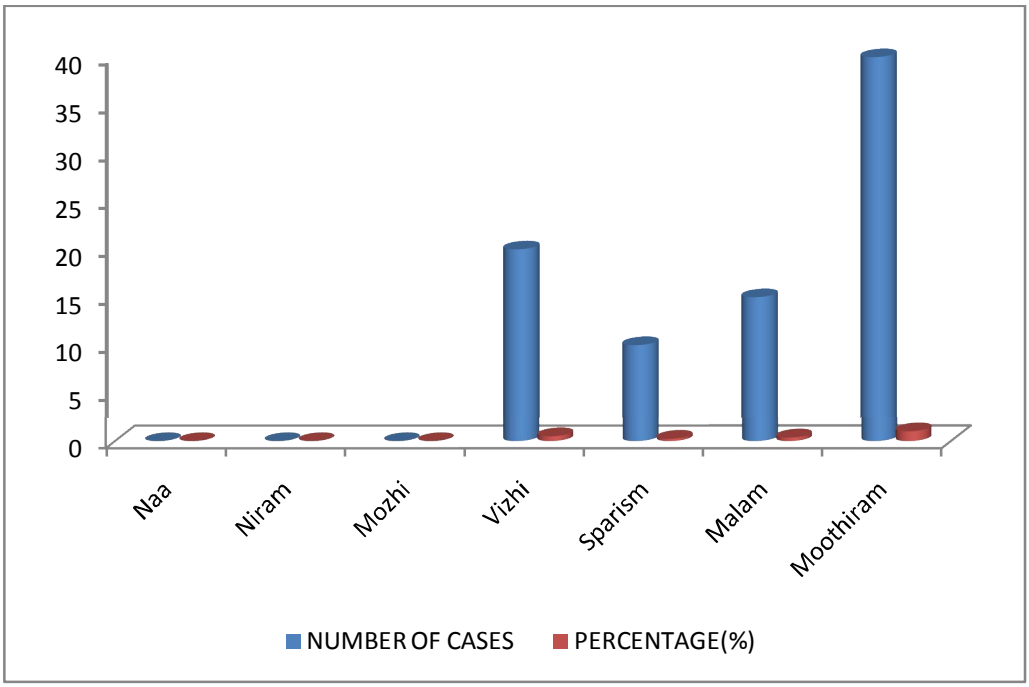
INFERENCE

Azhal vali was affected in 50% of cases .vali azhal was affected in 37.5% of cases and iyya azhal in 12.5% of cases.

11. ENN VAGAI THERUVUGAL

ENNVAGAI THERUVUGAL	NUMBER OF CASES	PERCENTAGE(%)
Naa	-	-
Niram	-	-
Mozhi	-	-
Vizhi	20	50%
Sparism	10	25%
Malam	15	37.5%
Moothiram	40	100%

ENN VAGAI THERUVUGAL



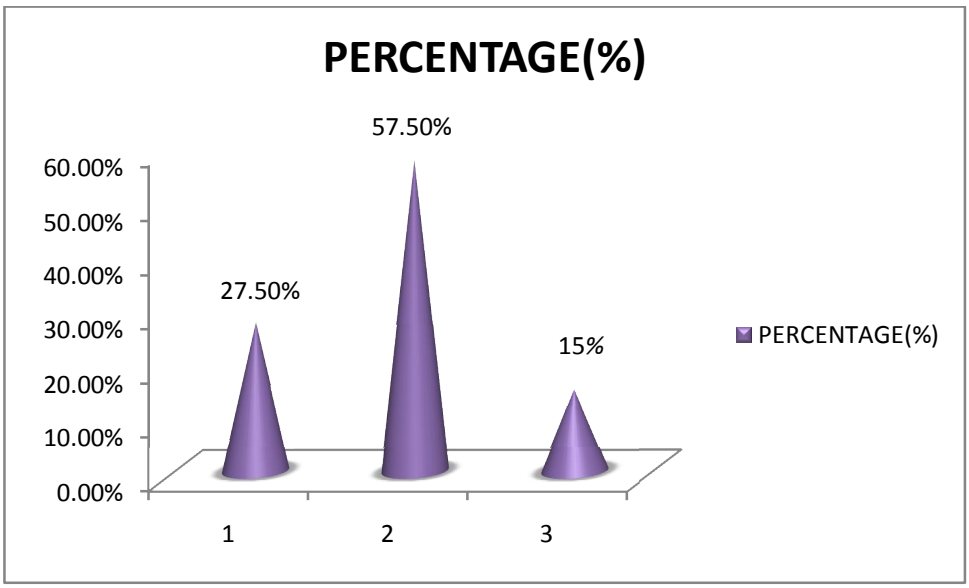
INFERENCE

Vizhi, Sparisam, Malam, Moothiram are affected.

12. NEIKURI

THATHU	NEIKURI	NUMBER OF CASES	PERCENTAGE(%)
Vatham	Spread like snake	11	27.5%
Pitham	Spread like ring	23	57.5%
Kabam	Spread like pearl	6	15%

NEIKURI



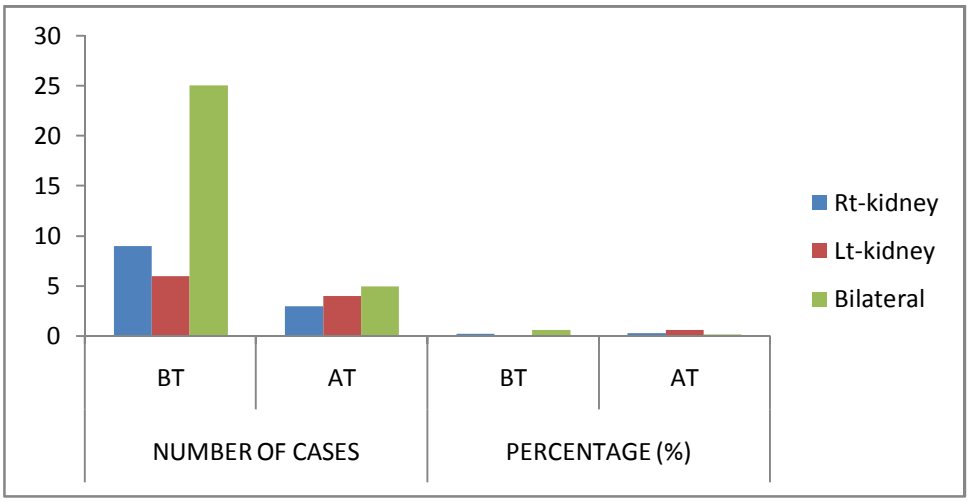
INFERENCE

57.5% cases were having pitha neikuri. 27.5% of cases were having vatha neikuri and 6% cases were having kaba neikuri.

13. DISTRIBUTION OF CALCULI BASED ON SIDE

SIDE	NUMBER OF CASES		PERCENTAGE (%)	
	BT	AT	BT	AT
Rt-kidney	9	3	22.5%	33.3%
Lt-kidney	6	4	7.5%	66.6%
Bilateral	25	5	62.5%	20%

DISTRIBUTION OF CALCULI BASED ON SIDE



INFERENCE

Before treatment:

Out of 40 patients 62.5% (25) were having bilateral renal calculi. 22.5% (9)were having Right renal calculi and 7.5% (6) were having left renal calculi.

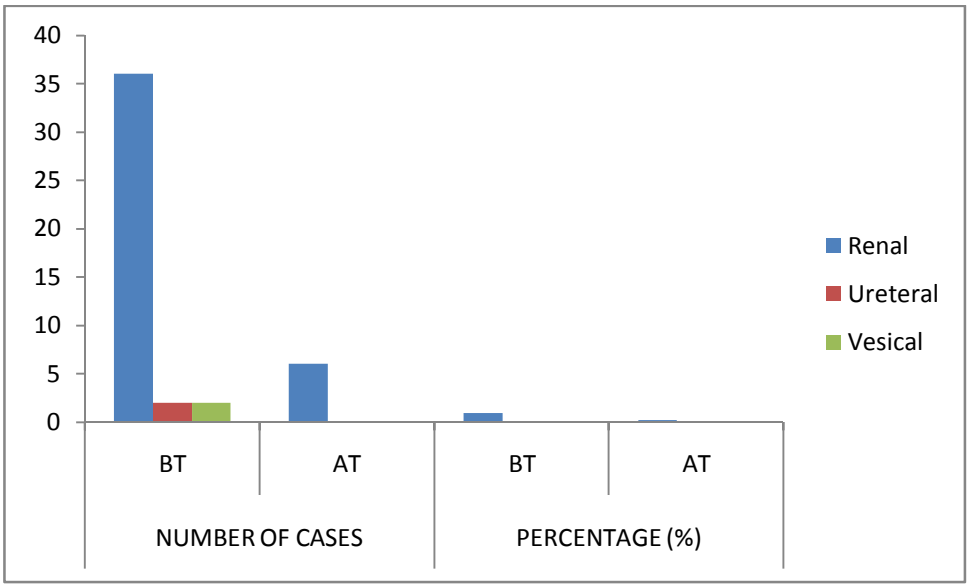
After treatment:

Out of 40 patients, after treatment 33.3% are having stones on RT kidney, 66.6% are having LT kidney stones, 20% are having bilateral stones.

14. DISTRIBUTION OF CALCULI BASED ON LOCATION

SIDE	NUMBER OF CASES		PERCENTAGE (%)	
	BT	AT	BT	AT
Renal	36	6	90%	16.66%
Ureteral	2	0	5%	0%
Vesical	2	0	5%	0%

DISTRIBUTION OF CALCULI BASED ON LOCATION



INFERENCE

Before Treatment

90% of cases were having Renal Stone, 5% were having Ureteral stones And 5% were having vesical stones.

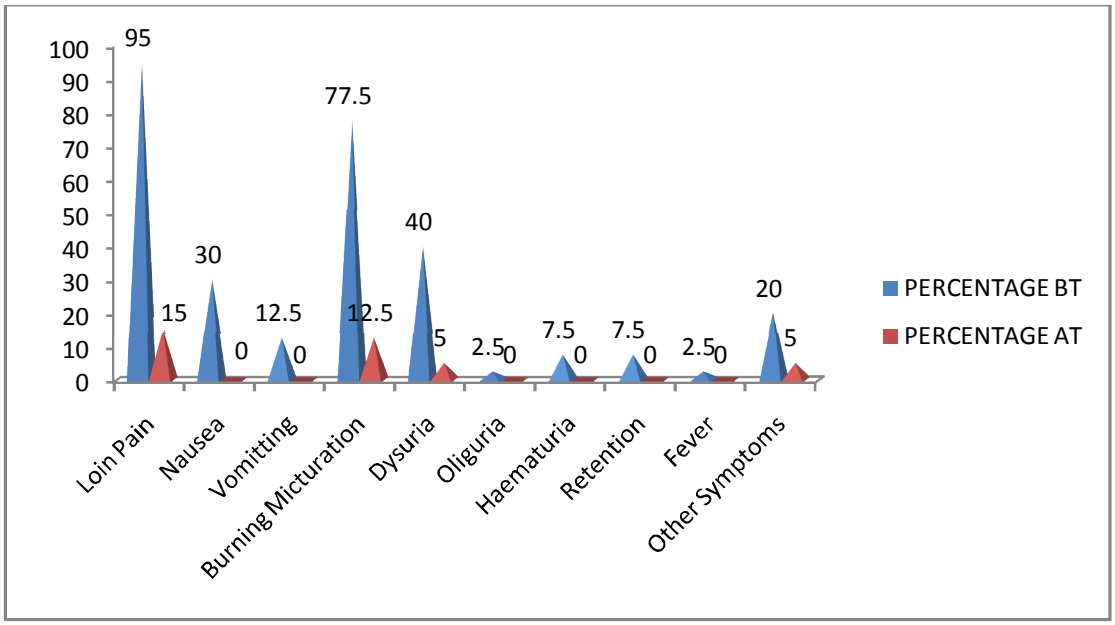
After Treatment

16.6% are having Renal stones, 0% are having ureteral stones, 0% having vesical stones.

15. CLINICAL MANIFESTATIONS

S.NO	SYMPTOMS	NUMBER OF CASES		PERCENTAGE	
		BT	AT	BT	AT
1	Loin Pain	38	6	95	15
2	Nausea	12	0	30	0
3	Vomitting	5	0	12.5	0
4	Burning Micturation	31	5	77.5	12.5
5	Dysuria	16	2	40	5
6	Oliguria	1	0	2.5	0
7	Haematuria	3	0	7.5	0
8	Retention	3	0	7.5	0
9	Fever	1	0	2.5	0
10	Other Symptoms	8	2	20	5

CLINICAL MANIFESTATIONS



INFERENCE

Before Treatment

95% had loin pain.77.5% had burning micturation.40% had dysuria.30% had nausea.

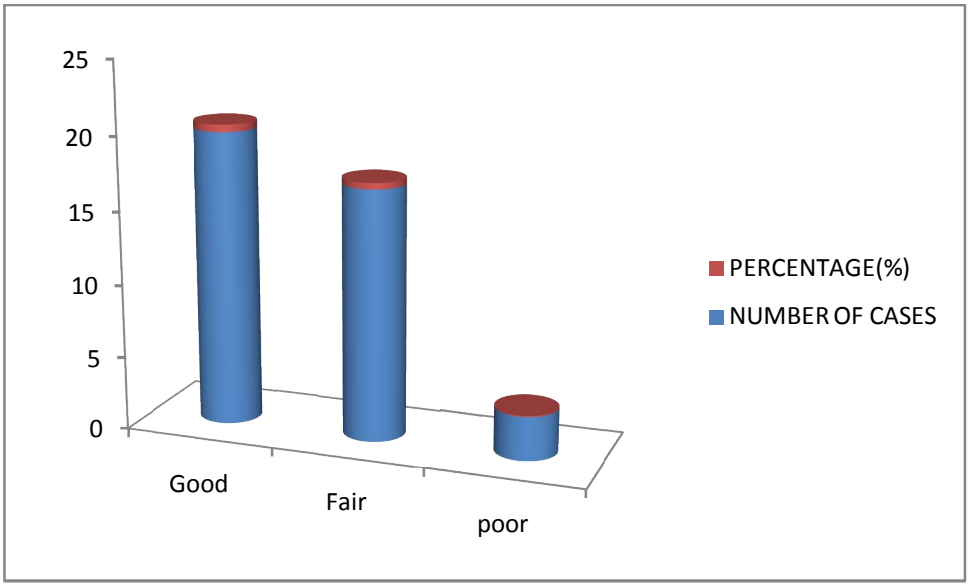
After Treatment:

15% had loin pain.12.5% had burning micturation.5% had dysuria.5% had nausea.

16. RESULTS AFTER TREATMENT

RESULT	NUMBER OF CASES	PERCENTAGE(%)
Good	20	50%
Fair	17	42.5%
poor	3	7.5%

RESULTS AFTER TREATMENT



INFERENCE

50% showed good results both by symptoms and sonographic findings. 42.5% showed fair results, relieved by their symptoms and 7.5% were not relieved from their problem.

LIST OF PATIENTS

S. NO	OP. No	NAME	AGE/ SEX	OCCUPATION	DURATION OF ILLNESS	DATE
1	7610	Senthilkumar	26/M	Mechanic	9-Months	13.12.2011
2	1136	Surendhar	46/M	Auto driver	5-Months	25.1.2012
3	3265	Kavitha	35/F	Housewife	8-Months	2.2.2012
4	4321	Ushanandhini	33/F	Housewife	6-Months	6.2.2012
5	646	Padmavathy	38/F	Housewife	4-Months	24.1.2012
6	7028	Perumal	21/M	Mechanic	3-Months	15.2.2012
7	4489	Sornambigai	41/F	Housewife	6-Months	13.2.2012
8	1590	Shyamala	40/F	Housewife	4-Months	2.3.2012
9	8228	Rajendran	38/M	Mechanic	6-Months	11.1.2012
10	8227	Natarajan	43/M	Mechanic	6-Months	11.1.2012
11	9845	Velappan	29/M	Student	5-Months	19-12-2011
12	7310	Ramesh	32/M	Merchant	3-Months	4.9.2012
13	1650	Sureshbabu	32/M	Mechanic	5-Months	9.4.2012
14	3288	Pandurangan	31/M	Teacher	4-Months	8.3.2012
15	1656	Anusha	24/F	IT-Eng	6-Months	2.3.2012
16	9539	Balaji	20/M	Student	2-Months	22.6.2012
17	4553	Deenadhayalan	48/M	BSNL-Optr	6-Months	5.6.2012
18	4517	Megala	43/F	Housewife	2-Months	12.7.2012
19	306	Beermaideen	48/M	Mechanic	7-Months	26.6.2012
20	5717	Karpagam	29/F	Housewife	4-Months	11.6.2012
21	9753	Johnmartin	60/M	Office work	3-Months	2.8.2012
22	5930	Sridhar	31/M	Business	4-Months	19.3.2012
23	9776	Jaganathan	55/M	Office Work	6-Months	1.8.2012
24	285	Azharudeen	25/M	Mechanic	7-Months	26.6.2012
25	7368	Santhosh	37/M	Htl Manager	6-Months	23.7.2012
26	958	Radhakrishnan	48/M	Type writer	3-Months	28.6.2012
27	6115	Pushparaj	60/M	Comp. Engr	4-Months	12.6.2012
28	4328	Yegiyakhan	38/M	Mechanic	5-Months	4.6.2012
29	6605	Ponpandiyan	22/M	Student	3-Months	19.6.2012
30	5763	Balasubramani	40/M	Teacher	6-Months	17.7.2012
31	6323	Madhan	20/M	Student	4-Months	13.6.2012
32	4155	Elumalai	30/M	Mechanic	7-Months	12.3.2012
33	4826	Dhachayini	45/F	Housewife	5-Months	13.7.2012
34	6603	Venkatesan	47/M	Electrician	6-Months	14.6.2012
35	430	Annadurai	37/M	Mechanic	3-Months	28.2.2012
36	2358	Kennadi raja	44/M	Teacher	7-Months	13.8.2012
37	4216	Parasivam	42/M	Business	3-Months	3.10.2012
38	6608	Ganesh	33/M	Merchant	4-Months	12.10.2012
39	7788	Senthil	32/M	Mechanic	6-Months	17.10.2012
40	4572	Ramani	48/F	House wife	4-Months	4.10.2012

RESULTS OF 40 KALLADAIPPU PATIENTS BEFORE & AFTER TREATMENT, GOVT. SIDDHA MEDICAL COLLEGE, ARIGNAR ANNA HOSPITAL, CHENNAI-106

S. No.	Name of the patient Age/sex	OP No.	Date of Treatment Started	Duration of Medicine in take	Size of stone BT	Whether stone expelled or not AT	Remarks
1	Senthilkumar 26/M	7610	13-12-2011	8-weeks	Bilateral RT 1cm LT 0.7cm	USG Normal	Completed
2	Surendhar 46/M	1136	25-01-2012	7-Weeks	Bilateral RT 8mm LT 6mm	One Stone expelled, LT3mm	Symptoms reduced
3	Kavitha 35/F	3265	02-02-2012	7-Weeks	Bilateral 6mm	USG Normal	Symptoms Relieved
4	Ushanandhini 33/F	4321	06-02-2012	7-Weeks	Bilateral 5mm	USG Normal	Completed
5	Padmavathy 38/F	646	24-01-2012	7-Weeks	Bilateral 4mm	USG Normal	Completed
6	Perumal 21/M	7028	15-02-2012	7-Weeks	RT 4mm	RT 4mm	Advise surgery
7	Sornambigai 41/F	4489	13-02-2012	7-Weeks	RT 2 Stones (4mm)	USG Normal	Symptoms Relieved
8	Shyamala 40/F	1590	2-03-2012	7-Weeks	Bilateral 5mm	RT 3mm	Symptoms Relieved
9	Rajendran 38/M	8228	11-01-2012	7-Weeks	LT 3.4mm	USG Normal	Completed
10	Natarajan 43/M	8227	11-01-2012	7-Weeks	RT 2 (4.3mm)	USG Normal	Completed
11	Velappan 29/M	9845	19-12-2011	7-Weeks	Bilateral gravel	USG Normal	Completed
12	Ramesh 33/M	7310	4-9-2012	7-Weeks	Bilateral RT 4mm LT 10mm,8.4mm	One stone expelled, Bilateral 3mm	Advised to continue Medicine.
13	Sureshbabu 32/M	1650	9-04-2012	7-Weeks	RT 4mm	RT 4mm	Symptoms reduced
14	Pandurangan 31/M	3288	8-03-2012	7-Weeks	LT 4.8mm	LT3mm	Completed
15	Anusha 24/F	1656	2-03-2012	7-Weeks	LT 6mm	USG Normal	Completed

S. No.	Name of the patient Age/sex	OP No.	Date of Treatment Started	Duration of Medicine in take	Size of stone BT	Whether stone expelled or not AT	Remarks
16	Balaji 20/M	9539	22-06-2012	7-Weeks	Bilateral RT 5mm LT 6mm	Bilateral 3mm	Symptoms Relieved
17	Venkatesan 47/M	6603	14-06-2012	7-Weeks	Bilateral 4mm	USG Normal	Completed
18	Deenadhayalan 48/M	4553	5-06-2012	7-Weeks	Bilateral 4mm	USG Normal	Completed
19	Megala 43/F	4517	12-07-2012	6-Weeks	LT 10mm	LT 3mm	Symptoms relieved
20	Beermaideen 48/M	306	26-06-2012	6-Weeks	RT 8mm	RT2mm	Completed
21	Karpagam 29/F	5717	11-06-2012	7-Weeks	Bilateral 4mm	USG Normal	Completed
22	Johnmartin 60/M	9753	2-08-2012	7-Weeks	Bilateral RT5mm LT 6mm	Bilateral 2mm	Symptoms relieved
23	Sridhar 31/M	5930	19-03-2012	7-Weeks	RT 8mm	USG Normal	Completed
24	Jaganathan 55/M	9776	01-08-2012	7-Weeks	LT 1.8cm	LT 1cm	Symptoms reduced
25	Azharudeen 25/M	285	26-06-2012	7-Weeks	Bilateral LT 6mm RT 6mm	Stone expelled	Completed
26	Santhosh 37/M	7368	23-07-2012	7-Weeks	RT 6mm	USG Normal	Completed
27	Radhakrishnan 48/M	958	28-06-2012	7-Weeks	Bilateral RT 7mm LT 7mm	RT 3mm	Symptoms relieved
28	Pushparaj 60/M	6115	12-06-2012	5-Weeks	Bilateral 6mm	USG Normal	Completed
29	Yegiyakhan 38/M	4328	04-06-2012	5-Weeks	Bilateral RT 5mm LT6mm	USG Normal	Completed
30	Ponpandiyan 22/M	6605	19-06-2012	7-Weeks	Bilateral RT4.6mm LT 5.9mm	Bilateral 3mm	Symptoms reduced
31	Balasubramani 40/M	5763	17-07-2012	7-Weeks	Bilateral RT10mm LT 5mm	Bilateral 3mm	Symptoms reduced

S. No.	Name of the patient Age/sex	OP No.	Date of Treatment Started	Duration of Medicine in take	Size of stone BT	Whether stone expelled or not AT	Remarks
32	Ramani 48/F	4572	4-10-2012	7-Weeks	RT 5mm,LT 4mm	USG Normal	Completed
33	Madhan 20/M	6323	13-06-2012	5-Weeks	LT 2(3.5mm)	USG Normal	Completed
34	Annadurai 37/M	430	28-2-2012	6-Weeks	Bilateral gravel	USG Normal	Completed
35	Elumalai 30/M	4155	12-03-2012	7-Weeks	RT 5.5mm	USG Normal	Completed
36	Kennadi Raja 44/M	2358	13-08-2012	7-Weeks	Bilateral RT 5mm LT 6mm	USG Normal	Completed
37	Parasivam 42/M	4216	3-10-2012	7-Weeks	1cm stone in the bladder	Stone expelled	Completed
38	Ganesh 33/M	6608	12-10-2012	6-Weeks	RT 5mm,4mm	USG Normal	Completed
39	Dhachayini 45/F	4826	13-07-2012	5-Weeks	Bilateral RT 3mm LT 3mm	USG Normal	Completed
40	Senthil 32/M	7788	17-10-2012	6-Weeks	Bilateral 5mm	USG Normal	Completed

LABORATORY INVESTIGATION REPORT

SL. No.	O.P. No.	Name	Age/Sex	*HAEMATOLOGICAL* REPORT																*URINE* ANALYSIS					
				BEFORE TREATMENT				AFTER TREATMENT				ESR (mm)				Hb (Gm)		Urea		Creatinine		BT			AT		
				TC CU/ mm	DC%			TC CU/ m m				BT		AT		BT	AT	BT	AT	BT	AT	Alb	Sug	Dep	Alb	Sug	Dep
					P	L	E		P	L	E	*1/2* hr	*1* hr	½ hr	1 hr												
1.	7610	Senthilkumar	26/M	9000	55	38	7	10500	62	34	4	22	45	15	30	9.0	12.5	23	15	.6	.5	+	NIL	Opc	NIL	NIL	NIL
2.	1136	Surendhar	46/M	9400	59	36	5	10100	62	34	4	5	12	4	7	11.8	13.5	26	15	.8	.4	+	NIL	Opc	NIL	NIL	NIL
3.	3265	Kavitha	35/F	10100	57	30	13	9600	53	39	8	10	22	7	15	9.0	13.4	29	15	.5	.4	+	NIL	Rbc	NIL	NIL	NIL
4.	4321	Ushanandhini	33/F	9000	57	39	4	9800	60	36	4	5	11	5	10	13.0	15.0	28	15	.2	.2	+	NIL	Opc	NIL	NIL	NIL
5.	646	Padmavathy	38/F	10200	62	33	5	10000	52	33	5	15	38	10	22	10.4	12.5	21	15	.4	.5	+	NIL	Opc	NIL	NIL	NIL
6.	7028	Perumal	21/M	9400	59	36	5	9800	60	36	4	15	5	11	30	12.4	15.0	24	15	.6	.4	+	NIL	Opc	NIL	NIL	NIL
7:	4489	Sornambigai	41/F	7800	56	38	6	10100	60	36	4	2	7	5	10	8.0	11.5	26	15	.8	.5	+	NIL	Rbc	NIL	NIL	NIL
8.	1590	Shyamala	40/F	9800	59	36	5	10200	60	34	6	3	6	5	10	16.0	12.4	28	15	.5	.4	+	NIL	Opc	NIL	NIL	NIL
9.	8228	Rajendran	38/M	9S0O	59	37	4	8900	55	41	4	15	20	5	12	13.0	12.0	29	15	.2	.2	+	NIL	Opc	NIL	NIL	NIL
10.	8227	Natarajan	43/M	10200	60	34	6	10400	60	36	4	5	11	25	60	13.0.	12.5	30	15	.4	.4	+	NIL	Epc	NIL	NIL	NIL
11	9845	Velappan	29/M	10400	60	34	6	9600	50	45	5	8	25	12	25	13.0	15.0	32	20	.6	.5	+	NIL	Opc	NIL	NIL	NIL
12.	7310	Ramesh	32/M	9100	57	38	5	9800	60	34	6	5	20	15	33	11.2	8.0	26	20	.8	.4	+	NIL	Opc	NIL	NIL	NIL
13.	1650	Sureshbabu	32/M	9700	57	38	5	9200	55	41	4	5	12	5	11	12.0	12.0	20	20	.5	.4	+	Nil	Epc	NIL	NIL	NIL
14.	3288	Pandurangan	31/M	9800	57	39	4	9400	57	38	5	12	13	10	18	10.0	13.4	29	20	.2	.2	+	NIL	Opc	NIL	NIL	NIL
15.	1656	Anusha	24/F	9700	59	33	8	9700	69	33	8	5	13	8	18	12.6	12.6	30	20	.4	.4	+	NIL	Opc	NIL	NIL	NIL
16.	9539	Balaji	20/M	10000	57	38	5	9000	54	42	4	5	12	5	7	16.0	13.0	32	25	.6	.5	+	NIL	Rbc	NIL	NIL	NIL
17.	4553	Deenadayalan	48/M	9000	53	40	7	10200	61	35	4	9	25	7	12	10.0	12.0	28	25	.8	.5	+	NIL	Opc	NIL	NIL	NIL
18.	4517	Megala	43/F	10400	66	28	6	10100	60	36	4	7	16	10	15	12.0	14.5	26	25	.5	.4	+	NIL	Epc	NIL	NIL	NIL
19.	306	Beermaideen	48/M	9000	54	42	4	9800	56	41	3	2	8	5	11	12.0	15.0	24	25	.2	.2	+	NIL	Opc	NIL	NIL	NIL
20.	5717	Karpagam	29/F	9400	55	41	4	9500	61	35	1	2	4	5 '	10	12.5	14.0	25	25	.4	.4	+	NIL	Opc	NIL	NIL	NIL
21.	9753	John Martin	60/M	9000	55	38	7	10500	62	34	4	22	45	15	30	9.0	12.5	27	20	.6	.5	+	NIL	Opc	NIL	NIL	NIL
22.	5930	Sridhar	31/M	9400	59	36	5	10100	62	34	4	5	12	4	7	11.8	13.5	26	20	.8	.6	+	Nil	Opc	NIL	NIL	NIL
23.	9776	Jagannathan	55/M	10100	57	30	13	9600	53	39	8	10	22	7	15	9.0	13.4	21	20	.5	.5	+	NIL	Rbc	NIL	NIL	NIL

SL. No.	O.P. No.	Name	Age/Sex	*HAEMATOLOGICAL* REPORT																*URINE* ANALYSIS					
				BEFORE TREATMENT				AFTER TREATMENT				ESR (mm)				Hb (Gm)		Urea		Creatinine		BT			AT		
				TC CU/ mm	DC%			TC CU/ m m				BT		AT		BT	AT	BT	AT	BT	AT	Alb	Sug	Dep	Alb	Sug	Dep
					P	L	E		P	L	E	*1/2* hr	*1* hr	½ hr	1 hr												
24.	285	Azharudeen	25/M	9000	57	39	4	9800	60	36	4	5	11	5	10	13.0	15.0	30	20	.2	.2	+	NIL	Opc	NIL	NIL	NIL
25.	7368	Santhosh	37/M	10200	62	33	5	10000	52	33	5	15	38	10	22	10.4	12.5	32	20	.4	.4	+	NIL	Opc	NIL	NIL	NIL
26.	958	Radhakrishnan	48/M	9400	59	36	5	9800	60	36	4	15	5	11	30	12.4	15.0	34	18	.6	.5	+	NIL	Opc	NIL	NIL	NIL
27.	6115	Pushparaj	60/M	7800	56	38	6	10100	60	36	4	2	7	5	10	8.0	11.5	25	18	.8	.4	+	NIL	Rbc	NIL	NIL	NIL
28.	4328	Yegiyakhan	38/M	9800	59	36	5	10200	60	34	6	3	6	5	10	16.0	12.4	27	18	.5	.4	+	NIL	Opc	NIL	NIL	NIL
29.	6605	Ponpandiyan	22/M	9S0O	59	37	4	8900	55	41	4	15	20	5	12	13.0	12.0	20	18	.2	.2	+	NIL	Opc	NIL	NIL	NIL
30.	5763	Balasubramani	40/M	10200	60	34	6	10400	60	36	4	5	11	25	60	13.0.	12.5	28	18	.4	.4	+	NIL	Epc	NIL	NIL	NIL
31.	6323	Madhan	20/M	10400	60	34	6	9600	50	45	5	8	25	12	25	13.0	15.0	26	18	.6	.5	+	Nil	Opc	NIL	NIL	NIL
32.	4155	Elumalai	30/M	9100	57	38	5	9800	60	34	6	5	20	15	33	11.2	8.0	24	18	.8	.6	+	NIL	Opc	NIL	NIL	NIL
33.	4826	Dhachayini	45/F	9700	57	38	5	9200	55	41	4	5	12	5	11	12.0	12.0	25	18	.5	.4	+	NIL	Epc	NIL	NIL	NIL
34.	6603	Venkatesan	47/M	9800	57	39	4	9400	57	38	5	12	13	10	18	10.0	13.4	27	18	.2	.2	+	NIL	Opc	NIL	NIL	NIL
35.	430	Annadurai	37/M	9700	59	33	8	9700	69	33	8	5	13	8	18	12.6	12.6	26	17	.4	.4	+	NIL	Opc	NIL	NIL	NIL
36.	2358	Kennadiraja	44/M	10000	57	38	5	9000	54	42	4	5	12	5	7	16.0	13.0	28	17	.6	.5	+	NIL	Rbc	NIL	NIL	NIL
37.	4210	Paramasivam	42/M	9000	53	40	7	10200	61	35	4	9	25	7	12	10.0	12.0	26	17	.8	.5	+	NIL	Opc	NIL	NIL	NIL
38.	6608	Ganesh	33/M	10400	66	28	6	10100	60	36	4	7	16	10	15	12.0	14.5	24	15	.5	.4	+	NIL	Epc	NIL	NIL	NIL
39.	7788	Senthil	32/M	9000	54	42	4	9800	56	41	3	2	8	5	11	12.0	15.0	25	15	.2	.2	+	NIL	Opc	NIL	NIL	NIL
40.	4572	Ramani	48/F	9400	55	41	4	9500	61	35	1	2	4	5	10	12.5	14.0	27	15	.4	.4	+	NIL	Opc	NIL	NIL	NIL

TC – Total Count, Hb – Hemoglobin, Dc – Differential Count, P – Polymorph, L – Lymphocyte, E – Eosinophil, ESR – Erythrocyte Sedimentation Rate, OEC – Occasional Epithelial Cells, OPC – Occasional Pus Cells, Rbc – Eed blood cell, Alb – Albumin, Sug – Sugar, Dep - Deposits

EXPELLED STONES

Name: Azharudeen – 25 / M

Name: Paramasivam – 42 / M

Name: Ramesh – 33 / M

Name: Surendar – 46 / M

Arignar Anna Government Hospital
of Indian Medicine
Arumbakkam, Madras-600 106

4/6/2012

Mrs. Karpagam 29/F OP No 4184 Siddha

USG whole abdomen

Liver - (N)

GB - (N)

Pancreas - Retroperitoneum - obscured by bowel gas

spleen (N)

Kidneys - B/o calculus ~ 3.5 mm noted (B/o)

in midpole of right kidney. E/o two calculi ~ 3.5mm noted in midpole of left kidney. E/o calculus ~ 3.8mm noted in lower pole of left kidney.

Urbladder - (N)

Uters - (N)

(R) Ovary ms ~ 28x24x21mm (vol ~ 7ml)

(L) ovary ms ~ 31x31x20mm (vol ~ 10mm)

Both ovaries appear polycystic.

Impr: - Polycystic ovaries
- Bil. renal calculi.
- Sug. clinical correlation — Dr

GEMINI
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360/2, Poonamallee High Road,
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CONSULTANT RADIOLOGISTS

Dr. G. Godwin, M.D., DNB | Dr. V. Ramkumar, DMRD, DNB | Dr. R. Chitrah, MD | Dr. V. Senthil, DMRD, DNB

Name : Mrs.Karpagam
Age : 29 Y/F
Ref.By.: Dr.R.Sathyavathy.,

Date : 06.10.2012
Id.No.: AK12/9462

Usg KUB & Pelvis

Kidneys:
RT.Kidney measures 9.4 x 4.0cms. LT.Kidney measures 9.6 x 4.1cms.
Corticomedullary differentiation is maintained on both sides.
Pelvicalyceal system on both sides appears normal.
No calculus is seen on either side.

Bladder:
Is partially filled.

(Transvaginal)

Uterus:
Anteverted and measures 8.1 x 4.3 x 5.3cms.
Myometrium shows normal echogenicity.
Endometrium is regular and measures 6.8mms.
Multiple nabothian cysts are seen in cervix.

Ovaries:
Right ovary measures 3.9 x 2.0 x 3.5cms. Volume 14.2cc.
Left ovary measures 4.0 x 1.9 x 3.3cms. Volume 12.8cc.
Both ovaries are enlarged and show multiple small follicles in the periphery measuring 4 to 6mms.

P.O.D:
P.O.D. is free.
No adnexal mass lesion is seen.

Impression:

- **Multiple nabothian cysts in cervix – Cervicitis.**
- **Polycystic ovaries.**
- **Normal Sonographic Study of Both kidneys, Uterus and Adnexa.**

Dr.R.Chitrah Sivan.,MD.,
Consultant Radiologist.
Ph.No:98848 15192.

Pragathi Scaning Centre

Dr. Sharath K. Shetty, M.B.B.S., M.D., (RD)
Consultant Radiologist & Sonologist

ABDOMINO - PELVIC ULTRASONOGRAPHY REPORT

NAME :	ASHIRUDDIN	AGE/SEX:	25 / Male
REF BY:	DEEKSHA HOSPITAL	DATE:	22/06/2012

LIVER : IS NORMAL IN SIZE. NORMAL ECHOTEXTURE. NO FOCAL LEISIONS. INTRA HEPATIC BILIARY RADICLES NORMAL. PORTAL VASCULATRE & CBD ARE NORMAL.

GALL BLADDER: SHOWS NO ABNORMAL INTRA LUMINAL ECHOES. NO PERI CHOLECYSTIC OEDEMA.

PANCREAS: IS NORMAL IN SIZE. NORMAL ECHOTEXTURE. NO M P D DILATATION.

SPLEEN: IS NORMAL IN SIZE. NORMAL ECHOTEXTURE.

RT KIDNEY : SHOWS MID POLE CALYCEAL CALCULUS MEASU . 6 mm.
CORTICO MEDULLARY. DIFFERENTIATION IS MAINTAINED.
R K : L : 101 mm PARENCHYMAL THICKNESS : 12 mm

LT KIDNEY : SHOWS MILD DEGREE OF HYDROURETRONEPHROSIS SECONDARY TO Approx. 6 mm. CALUCLUS MIDDLE ONE THIRD OF URETER. CORTICO MEDULLARY. DIFFERENTIATION IS MAINTAINED.
L K : L : 103 mm PARENCHYMAL THICKNESS : 14 mm

RIF : NO MASS / ABCESS / COLLECTION. BOWEL MOVEMENT NORMAL.

OTHERS: NO ASCITIES / PLEURAL EFFUSION / NO OBVIOUS R/P NODES.

URINARY BLADDER: NO ABNORMAL INTERNAL ECHOES. NORMAL WALL THICKNESS.

PROSTATE : NORMAL IN SIZE. NORMAL ECHOTEXTURE.

IMPRESSION : * LEFT SIDED MILD DEGREE OF HYDROURETRONEPHROSIS SECONDAR TO Approx. 6 mm. CALUCLUS MIDDLE ONE THIRD OF URETER.
* NON OBSTRUCTIVE RIGHT RENAL MID POLE CALYCEAL CALCULUS MEASU : 6 mm.

SUGGESTED REVIEW SCAN AFTER FLUSH THERAPHY.

DR : K.S. SHETTY . M.D (R D)., RADIOLOGIST & SONOLOGIST.

1303, Y.M. Gopalappa Building, Opp. BDK Complex, Next to Anjaneya Temple, Santhe Circle, B.B. Road, Yelahanka Old Town, Bangalore - 560 064. **Ph. : 080-4173 3346 / 988 097 6773**

SWAMI VIVEKANANDA DIAGNOSTIC CENTRE

Lions Edifice for Service Trust Complex,D.G.Vaishnav College

Chennai-600106,PH:044-43853101,43853102

Patient name	MR. AZARUDEEN	Age/Sex	25 Years / Male
Patient ID	03254	Visit no	1
Referred by	Dr. SATHIYAVATHY	Visit date	17/09/2012

Abdomen and KUB Scan Report

Abdomen

Liver filled with homogeneous parenchymal echoes. No abscess or mass lesion in the liver

Gall bladder appeared normal. No calculi seen in the gall bladder

Commonduct appeared normal. No calculi seen in the commonduct.

Pancreas appeared normal

Spleen measured 11.5 cms.

Spleen appeared normal

Aorta appeared normal. No para aortic nodes seen.

Peritoneal cavity appeared normal

KUB

Right kidney measured 10.2 X 4.0 cms.

Cortex and collecting system of right kidney appeared normal. No calculi seen.

Left kidney measured 10.4 X 4.3 cms.

Cortex and collecting system of left kidney appeared normal. No calculi seen.

Bladder appeared normal

Prostate measured 3.6 X 3.2 X 2.9 cms. (Weight = 17.4 gms.)

Prostate appeared normal. No intra vesical enlargement of prostate gland seen.

Impression

Normal study.

DR.S.KAVITHA, M.B.B.S.,
SONOLOGIST
REGN. No. 70356

DR.S.KAVITHA
SONOLOGIST

DISCUSSION

'KALLADAIPPU' is a common disease pertaining to the kidney. Large population are suffering from this disease. But they are not completely relieved from their symptoms by other systems of medicine. Hence with the help of trial medicine from siddha system, results and observations are noted for this study.

The patients were examined based on siddha and as well as modern aspects. All the necessary investigations were made during the study. The results obtained from their studies were discussed below for better conclusion.

Trial medicine administered was,

Megarajanga chooranam-1gm2 times/day

With butter after food for 48 days.

1.Sex distribution:

Among 40 cases 30 were males and 10 were females.

2.Age distribution:

Although all the decades of people are affected 37.5% were affected in 3rd decade, 25% were in 2nd decade.

3.Occupation:

Mixed categories of people are affected, from housewife to working woman, students to retired person. Among the total, 30 males, 20 are mechanics and office workers. In overall occupational distribution also they occupy the first place and next comes manual workers and students.

4.Food Habits:

All of my patients were having mixed dietary habits, eat both vegetarian and non vegetarian food.

5.According to season:

The highest incidence were noted in Elavenir Kaalam and 22.5% cases were noted in munpani kaalam. This shows that due to hot climate, majority of the people have been reported during this period.

6.Distribution of Thinai

According to thinai the highest distribution 80% was noted in neithal and 20% was noted in Kurinchi.

7.On clinical manifestations

All of my patients were present one or more urinary symptoms, 95% were having loin pain, 77.5% were having burning micturition, 40% had dysuria, 30% had nausea, 12.5% had vomiting, 7.5% had haematuria and 1 patient had fever and retention.

8.Mukkutram:

Disturbance of vatham:

Among the patients 100% were affected in Abanan and Viyanan, 32.5% in Uthaman, 20% in samanan and 30% in Kirukaran.

Affected Abanan produce burning micturition, constipation and haematuria.

Affected viyanan produced tenderness from loin and groin.

Affected samanan produce loss of appetite, indigestion and flatulence.

Affected uthaman produce nausea, vomiting

Affected koorman produce nausea

Disturbance of pitham:

Among the treated patients, 100% were affected in sathaga pitham, 22.5% were affected in ranjagapitham, 20% were affected in anarpitham.

The affected anarpitham produced loss of appetite. Affected Ranjagapitham produced pallor of skin, eye, nail and reduced haemoglobin.

Affected sathagapitham produce difficulty in doing our routine work.

Disturbance of kabam

7.5% were affected by pothagam. 20% were affected in Klethagam. 5% were affected by santhigam.

Distributed Klethagam produces loss of appetite. Affected santhigam produces low back pain, knee joint pain, affected pothagam produces decreased taste sensation.

9.Ezhu udal Thathukkal

In this heading 100% of patients are suffered from tiredness and 22.5% are having anemia.

10.Enn vagai Thervugal – NAADI

In this, on examination of naadi, 37.5% are having vathapitham naadi and 50% are having pithavatha naadi and 12.5% are having kabhapitha naadi. A seat anchor of Azhal is urinary bladder. The trial drugs balance the impaired azhal and treats the urinary symptoms.

11.Neikuri

On neikuri examination 57.5% were having pitha neer. 27.5% were having vatha neer and 15% were having kabaneer.

A drop of gingely oil dropped into the early morning urine sample in a bowl, may result in, spread like snake-called vatham, like a ring in pitham, like pearl in kabam.

12. Urinary calculi based on site

In total 40 patients 23 were having bilateral stones (57.5%), 22.5% right side stones and left side is 20%.

13. Urinary calculi based on location

Most of the patients 36 (90%) were reported with renal calculi and only 2 patients – 5% were with ureteric calculi and vesical calculi.

This shows that the ureteric calculi are easily pushed out by Allopathic treatments and the people with calculi in the kidney approaches siddha system for a permanent relief.

14. Special Investigation:

USG – abdomen, and pelvis is advised for all the patients, to confirm the diagnosis.

After confirming the diagnosis, the patients were given the trial medicine and instructed to follow the diet and other restrictions based on Siddhasystem.

15. Urine Analysis:

It was observed that, 80% of cases showed PUS cells in urine.

16. Mode of action of the drug

According to Siddha system.

Based on Suvai:

The trial medicine Megarajanga chooranam has sweet taste. This taste will mitigate the vitiated pitham which is the main cause for Kalladaippu. This medicine act against the vitiated pitham. So it is considered as Ethirurai Maruthuvam.

Based on Veeriyam: (Nature)

The trial medicine Megarajanga chooranam possess Thatpa Veeriyam. So it cures pitha diseases.

By this Megarajanga chooranam treats Kalladaippu Noi.

Based on Adjuvant action:

Megarajanga chooranam is given with Butter which corrects pitha dearrangement.

19. Phytochemical Analysis

Megarajanga chooranam has sulphide, copper, iron, potassium, magnesium, alkaloids, reducing sugar.

In this potassium, magnesium and sulphate have diuretic actions.

20. Toxicological analysis

Acute and subacute toxicity studies were conducted at Veltech College of Pharmacy. At the end of toxicity studies the animals were sacrificied and the hematological parameters (TC, DC and Hb), Biochemical parameters (LFT, KFT) and histopathology of vital organs like Liver, Kidney, Spleen and Lungs were carried out.

21. Pharmacological analysis

Pharmacological studies of the trial medicine Megarajanga chooranam showed Urolithiatic and diuretic actions in albino wistar rats.

The results of preclinical screening, the results of Chemical analysis, Toxicological studies, Pharmacological studies are shown in annexures.

22. Statistical Analysis

The preclinical studies of trial medicine Megarajanga chooranam statistically analysed and showed significant result.

Statistical analysis of clinical study were done for the subjective and objective parameter, observed before and after treatment statistical results of preclinical and clinical study were attached to annexure.

23. Results after treatment

Many of my patients were relieved of their problem, 50% showed good result that is both by symptoms and by X-Ray and sonographic findings, 42.5% showed fair result who are relieved of their symptoms but having stones sonographically, 3 patients that is 7.5% were not relieved of their problem.

SUMMARY

- I like to summarize this study with the following highlights.
- Males are more prone to get kalladaippu than females according to my studies.
- In age distribution, 3rd and 4th decades of people are more affected
- Housewifes and manual workers occupy the first two places in occupational classification
- All of my patients, had mixed dietary habits
- Higher incidence of cases were noted in Elavenir kaalam (April – June)
- In the disturbance of Ezhu udal thathukkal, 100% were affected by saaram, 22.5% were affected by senneer and 5% were affectedby Enbu.
- In Naadi, most of the patients were having pitha vadha naadi and vadhapitha naadi
- In Neikuri examination 57.5% were having pithaneer
- All of my patients were having urinary presentation, 95% were having loin pain, 77.5% had burning micturition, 40% had dysuria. 30% had nausea, 12.5% had vomiting, 7.5% had retention and haematuria.
- Most of the 40 patients, had stone in kidney (ie) Bothsides of urinary system is affected in majority of 57.5%
- All of my patients were administered with my trial medicine

Megarajanga chooranam – 1 gm bd with butter after food for a period of 48 days.

After treatment with this trial medicine most of the symptoms like loin pain, burning micturition and dysuria are relieved by the expulsion of the stone. And the trial medicine shows 62.5% good result.

CONCLUSION

- Kalladaippu is a common disorder of Pithakuttram. The dearranged pitham is settled down by the trial medicine having suvai Inippu thereby the medicine acts as Ethirurai maruthuvam to cure the disease.
- Most of the cases noted in kaarkalam, ilavenir kaalam in my clinical trial. So, people should take all preventive measures during this period and take enough water.
- Toxicological studies showed no acute or subacute toxicity.
- Pharmacological study reveals that the trial medicines possess diuretic as well as lithotriptic actions.
- During the clinical trial, no adverse reactions or complications were observed.
- The palatability of the trial drug is sweet, so it is easier to consume to the patients.
- The trial medicine Megarajanga chooranam showed good results with the expulsion of stone in few patients and relieve urinary symptoms in almost 90% of patients.
- Once again siddha medicine proves itself as a great boon to mankind.

ANNEXURE – 1

PHYTO - CHEMICAL ANALYSIS
CHEMICAL ANALYSIS OF TRIAL MEDICINES

Preparation of Sodium Carbonate extract

2gm of Trial medicine **Megarajanga choornam** - is weighed accurately and mixed with 5gm of sodium carbonate taken in a 100ml beaker and 20ml of distilled water is added. The solution is boiled for 10 minutes, cold and then filtered. The filterate is called sodium Carbonate extract.

S. NO	EXPERIMENT	OBSERVATION	INFERENCE
1.	**TEST FOR ACID RADICALS**		
a)	**Test for Sulphate** 2 ml of the above prepared extract is taken in a test tube. To this add 2 ml of 4% Ammonium oxalate solution.	-	-
b)	2 ml of extract is added with 2 ml of dilute Hydrocholoric acid until the effervescence ceases off. Then 2 ml of Barium chloride solution is added.	A white precipitate develops	Present
2.	**Test for Chloride** 2 ml of extract is added with dilute Nitric acid till the effervescence ceases. Then 2 ml of silver Nitrate solution is added.	Cloudy appearance develops	Presence of Chloride
3.	**Test for Phosphate** 2 ml of the extract is treated with 2 ml of Ammonium Molybdate solution and 2 ml of concentrated Nitric Acid.	Cloudy appearance develops	Presence of phosphate
4.	**Test for Carbonate** 2 ml of the extract is treated with 2 ml of Magnisium Sulphate solution.	-	-
5.	**Test for Sulphide** 1 gm of the substance is treated with 2 ml of concentrated Hydrocholric acid	Absent	Absent
6.	**Test for Nitrate** I gm of the substance is heated with copper turnings and concentrated Suophuric acid and viewed the test tube vertically down.	Absent	Absent
7a.	**Test for Fluoride and Oxalate** 2 ml of the extract is added with 2 ml of dilute Acetic acid and 2 ml of Calcium Chloride solution and heated.	Absent	Absent

S. NO	EXPERIMENT	OBSERVATION	INFERENCE
b.	5 drops of clear solution is added with 2 ml of dilute sulphuric acid and slightly wanned. To this, 1 ml of dilute Potassium Permanganate solution is added.	Present	Present
8.	**Test for Nitrite** 3 drops of the extract is placed on a filter paper. On that, 2 drops a Acetic Acid and 2 drops of Benzidine solution is placed.	Absent	Absent
9.	**Test for Borate** 2 pinches of the substance is made into paste by using Sulphuric acid and Alcohold (95%) and introduced into the blue flame.	Absent	Absent
II.	**TEST FOR BASIC RADICALS**		
10.	**Test for lead** 2 ml of the extract is added with 2 ml of Pottassium Iodide solution	Absent	Absent
11a	**Test for Copper** One pinch of substance is made into paste with concentrated Hydrochloric acid in a watch glass and introduced into the non luminous part of the flame.	Absent	Absent
b.	2ml of the extract is added with excess of Ammonia solution	Absent	Absent
12.	**Test for Aluminium** To the 2nd ml of extract. Sodium Hydroxide solution is added in drops to excess.	Absent	Absent
13a	**Test for Iron** To the 2nd ml of extract, 2 ml of Ammonium Thiocyanate solution is added.	Present	Present
b.	To the 2nd ml of extract, 2 ml of Ammonium Thiocyanate solution and 2 ml of concentrated Nitric Acid is added.	Present	Present
14.	**Test for Zinc** To the 2 ml of extract Sodium Hydroxide solution is added in drops to excess.	A white precipitate appears	Present
15.	**Test for Calcium** 2 ml of the extract is added with 2 ml of 4% Ammonium Oxalate solution.	Absent	Absent
16.	**Test for Magnesium** To 2ml of extract, Sodium Hydroxide solution is added in drops to excess.	A white precipitate appears	Present

S. NO	EXPERIMENT	OBSERVATION	INFERENCE
17.	**Test for Ammonium** To 2 ml of extract few ml of Nessler's Reagent and excess of Sodium Hydroxide solution are added.	-	-
18.	**Test for Potassium** A pinch of substance is treated with 2 ml of Sodium Nitrite solution and then treated with 2 ml of Cobal Nitrate in 30% glacial Acetic acid.	Yellow precipitate forms	Present
19.	**Test for Sodium** 2 pinches of the substance is made into paste by using Hydrocholoric acid and introduced into the blue flame.	Yellow color flame appears	Absent
20.	**Test for Mercury** 2 ml of the extract is treated with 2 ml of Sodium Hydroxide solution.	Absent	Absent
21.	**Test for Arsenic** 2 ml of extract is treated with 2 ml of silver Nitrate solution	Absent	Absent

The given sample contains

ACID RADICALS

1. Megarajanga Choornam
    - Sulphate
    - Phosphate

BASIC RADICALS

1. Megarajanga Choornam
    - Ferrous Iron
    - Copper
    - Potassium
    - Magnesium

Diuretics:

Potassium, Copper, magnesium and sulphide

Lithotriptic

Potassium and magnesium.

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S.No	Title of The Project	Name of The Investigator	Approval status/Remarks	Project Reference
4.	Evaluation of ovulation inducing activity for infertility and toxicological studies for Uppu parpam.	Dr. N. Kavitha	According to the protocol 36 rats were proposed, but while scrutinizing for pooling the final data, only 30 rats were sanctioned.	XIII/VELS/PCOL/ 04/2000/CPCSEA/I AEC/11.08.2012
5.	Hepatoprotective activity of Charaparpam by CCL4 induced method in rats	Dr. S. Umera	Total number of animals proposed was 60 rats. But 60 mice were sanctioned because, it was advised to share the control and standard group results. Since the similar pattern of the study has been planned in the same department, hence these data will serve as common.	XIII/VELS/PCOL/ 05/2000/CPCSEA/I AEC/11.08.2012
6.	A study on Poovarampattai kudineer choornam for the treatment of Swethakuttam.	Dr. A. Chinnaswamy	Total number of animals proposed was 42 rats. But only 36 animals were sanctioned.	XIII/VELS/PCOL/ 06/2000/CPCSEA/I AEC/11.08.2012
7.	A study of Kanthaga parpam for the treatment of kumbavatham.	Dr. G. Krishnaprakash	Total number of animals proposed was 36 rats and sanctioned.	XIII/VELS/PCOL/ 07/2000/CPCSEA/I AEC/11.08.2012
8.	Hypolipidemic activity of Kadukkai chooranam.	Dr. F. Priya	Total number of animals proposed was 48 rats, and it was advised to minimize the number to 40 rats only.	XIII/VELS/PCOL/ 08/2000/CPCSEA/I AEC/11.08.2012

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S.No	Title of The Project	Name of The Investigator	Approval status/Remarks	Project Reference
17.	Studies on Acute, Subacute toxicity – Diuretic activity of *Jalamanjari Chendooram.*	Dr. I. Nithya Mala	The candidate proposed 36 rats and 36 mice for the experimentation. But, experts suggested that the study data can be shared with other co workers. So, it is advised to minimize the number to 10 rats and 25mice were sanctioned.	XIII/VELS/PCOL/ 17/2000/CPCSEA/I AEC/11.08.2012
18.	Evaluation of Centrally acting Analgesic property and CNS Activity of Sathikkaipodi in mice.	Dr. P. Kavitha	Totally 35rats were proposed and sanctioned.	XIII/VELS/PCOL/ 18/2000/CPCSEA/I AEC/11.08.2012
19.	A Preclinical Trial Of *Megarajanga Choornam* For T Treatment Of Kalladaippu.	Dr. R. Sathyavathy	Totally 35rats were proposed and sanctioned.	XIII/VELS/PCOL/ 19/2000/CPCSEA/I AEC/11.08.2012
20.	Bronchodilator and lithotripic activity of *Neeradaipu thelineer*	Dr. Tamizh Magal	Total number of animals sanctioned was 50 rats and 13guinea pigs. Permitted to proceed. But it is advised to share the common group data with similar pattern of projects if possible.	XIII/VELS/PCOL/ 20/2000/CPCSEA/I AEC/11.08.2012
21.	Studies on Acute, Subacute toxicity – Diuretic activity of *Ashta gunma Thiraavagam.*	Dr. B. Kanimozhi	35 rats were Sanctioned and advised to reduce the animal groups from 8 to 5.	XIII/VELS/PCOL/ 21/2000/CPCSEA/I AEC/11.08.2012

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ANNEXURE - 2

ANTIUROLITHIATIC ACTIVITY OF MEGARAJANGA CHOORANAM IN ETHYLENE GLYCOL INDUCED LITHIATIC RATS

INTRODUCTION

Nephrolithiasis is common, affecting up to 10% of the population at some point during their lifetime. Calcium-containing stones are the most commonly occurring to an extent of 75-90% followed by magnesium ammonium phosphate to an extent of 10-15%, uric acid 3-10% and cystine 0.5-1%. Calcium oxalate stones are found in two different varieties, calcium oxalate monohydrate or calcium oxalate dihydrate. Calcium oxalate monohydrate, the thermodynamically most stable form, is observed more frequently in clinical stones than calcium oxalate dihydrate and it has a greater affinity for renal tubular cells, thus responsible for the formation of stones in the kidney.

Oxalate, a metabolic end product and a major constituent of the majority of renal stones, has been shown to be toxic to renal epithelial cells of cortical origin. It has been observed that exposure of renal epithelial cells to oxalate which is a constituent of most kidney stones leads to a disruption of the normal activities of the renal epithelial cells such as altered membrane surface properties and cellular lipids, changes in gene expression, disruption of mitochondrial function, formation of reactive oxygen species and decreased cell viability. Various mechanisms have been proposed to explain crystal retention. As a result of crystal growth and agglomeration, particles may be formed that are too large to freely pass the renal tubules. Alternatively, relatively small crystals could be retained by adhering to the surface of the urothelial lining and then increase in size. The surgical methods available to treat kidney stones like extracorporeal shock wave lithotripsy have serious side effects. Therefore, it is worthwhile to look for an alternative for the management of urolithiasis. Urolithiasis is the third most common disorder of the urinary tract, the others being frequently occurring urinary tract infections

and benign prostatic hyperplasia. The worldwide incidence of urolithiasis is quite high and in spite of tremendous advances in the field of medicine, there is no truly satisfactory drug for the treatment of renal calculi.

Siddha system plays a vital role in providing plant based medicines to overcome this disease. Kidney oxalate stone is the result of supersaturation of urine with certain urinary salts such as calcium oxalate. The supersaturation of urine with CaOX(Calcium oxalate), the most common component of kidney stones, is an important factor in crystallization, with later factors being nucleation, growth and aggregation.

Megarajanga Chooranam has been studied initially for diuretic property. Generally, drugs having diuretic activity are also known to have antiurolithiatic activity. Hence in the present study, an effort has been made to evaluate the antiurolithiatic activity of Megarajanga Chooranam using ethylene glycol induced lithiasis in rats.

MATERIALS AND METHODS

Preparation of drug and stock solution

The suspension of siddha drug Megarajanga Chooranamin 2%(w/v) CMC was prepared for oral administration by gastric intubation method.

Animal selection

For acute toxicity studies, Wistar albino mice of either sex weighing between 28 and 30 g were selected. For the antiurolithiatic study, male Wistar weighing between 180-220 g were used. The animals were acclimatized to standard laboratory conditions (temperature: 25±2°C) and maintained on 12-h light: 12-h dark cycle. They were provided with regular rat chow and drinking water ad libitum. (Approval number: XIII/VELS/PCOL/19/ 2000/CPCSEA/IAEC/ 08.08.2012).

Acute toxicity studies

The acute oral toxicity study was carried out as per the OECD guidelines 425. One-tenth of the median lethal dose was taken as an effective dose.

Ethylene glycol induced urolithiasis model

Ethylene glycol induced urolithiatic model in rat was be used to assess the effect of Megarajanga Chooranam. The study is designed to find out the effect of Megarajanga Chooranam on therapeutic usage against ethylene glycol induced urolithiasis. All rats were housed in metabolic cages for entire duration of the experiment. Animals were divided into five groups containing six animals in each. Group I served as control and received regular rat food and drinking water ad libitum. Ethylene glycol (0.75%) in drinking water was fed to Groups II-V for induction of renal calculi till 28th day. Group II received Ethylene glycol alone and served as urolithiatic control. Group III received standard antiurolithiatic drug, cystone (750mg/kg body weight) from 15th day till 28th day. Groups IV received Megarajanga Chooranam (50mg/kg body weight) from 15th day till 28thday, Group V received Megarajanga Chooranam(100mg/kg body weight) from 15st day till 28th day.

Group and Treatment

Group 1: Treated with Normal saline

Group 2: Treated with Control (ethylene glycol) + vehicle

Group 3: Treated with Standard (ethylene glycol + Cystone)

Group 4: Teated with Megarajanga Chooranam (50mg/kg) + ethylene glycol

Group 5: Treated with Megarajanga Chooranam(100mg/kg) + ethylene glycol

All doses were given once daily by oral route.

Assessment of antiurolithiatic activity

Collection and analysis of urine:

All animals were kept in individual metabolic cages and urine samples of 24h were collected on 28th day. Animals will be having free access to drinking water during the urine collection period. A drop of concentrated hydrochloric acid was added to the urine before being stored at 4°C. Urine was analyzed for calcium, phosphate and oxalate content.

Serum Analysis:

After the experimental period, blood was collected from the retro-orbital vein under anesthetic conditions and animals were sacrificed by cervical decapitation. Serum was separated by centrifugation at 10,000x g for 10 min and analyzed for creatinine, uric acid and urea nitrogen.

Kidney homogenate analysis:

The abdomen was cut open to remove both kidneys form each animal. Isolated kidneys were cleaned off extraneous tissue and preserved in 10% neutral formalin. The kidneys were dried at 80°C in a hot air oven. A sample of 100mg of the dried kidney were boiled in 10ml of 1N hydrochloric acid for 30min and homogenized. The homogenate was centrifuged at 2000x g for 10min and the supernatant was separated. The calcium, phosphate and oxalate content in kidney homogenate were determined.

DIURETIC ACTIVITY:

Standarsization Of Furosemide

Seven groups of six male wistar albino rats were employed four doses of 10,15,20,25-mg/kg b.w of furosemide were administered intraperitoneaaly to each group of rats separately. The control animals received normal saline alone. The animals were placed in separate cages and the urine output over 24hr period was collected. This procedure was repeated. The most consistent dose (15mg/kg b.w) was adapted for dosing.

Evaluation of diuretic activity

Five groups of six male Wistar albino rats were used. First group received normal saline. Second group received Megarajanga Chooranam 50mg/kg. The third group received Megarajanga Chooranam 100mg/kg. The fourth group was administered furosemide 20mg/kg. Immediately after administration of the drug, the rats were placed in metabolic cages, specially designed to separate urine and fecal matter and was observed at room temperature. The animals were denied for food and water during the experiment. The urine volume (ml/day) was measured and then assayed for Na^+ and K^+ and Cl^- concentrations in mMol/l, Cl was measured using routine method.

Statistical analysis:

Results expressed as mean ± S.E.M. Differences among data was determined using one-way ANOVA followed by Dunnet 't'test.

RESULTS AND DISCUSSION

Ethylene glycol induced urolithiasis resulted in significant elevation of urine and kidney calcium, oxalate, inorganic phosphate and serum blood urea nitrogen, creatinine, uric acid compared to normal control group. Treatment with cystone (750 mg/kg) and Megarajanga Chooranam reduced the bio-chemical changes induced by ethylene glycol. In order to probe the possible mechanism by which Megarajanga Chooranam cures renal damage caused by ethylene glycol, investigation on levels of various stone inhibitors like total protein, magnesium and citrate was studied. There was significant rise on total protein, magnesium and citrate after treatment with cystone and Megarajanga Chooranam.

Administration of ethylene glycol significantly reduced the body weight, urine volume and pH of urine as compared to normal group. Rats treated with cystone and Megarajanga Chooranam also showed significant decreased in body weight, urine volume and pH of urine as compared to control group. The histopathological study of the kidney sections also supported the above results.

In all the stone forming rats there was damage to the last part of the nephron, collecting system and peritubular interstitium as compared to the normal rat kidney architecture. The tubules appeared focally ecstatic and surrounded by inflammatory infiltration.

Flattened epithelium with focal vacuolar degeneration and single cell necrosis bordered the tubules, which focally contained hyaline casts. Inflammatory infiltration was mainly composed of mature lymphocytes infiltrating tubular epithelium. Irregular crystals were present inside the tubules and in the peritubular interstitium, along the nephron and at papillary level. The Megarajanga Chooranam treated groups showed normal histology of the kidney, and shows normal glomeruli, slight oedema of the tubular cells compared to standard drug treated animals. The kidneys excised from ethylene glycol treated group were larger and heavier than from the control animals. When observed under light microscope, many crystalline deposits in the histological preparations were seen in tubules of all regions of kidney.

In Megarajanga Chooranam along with EG treated rats, such deposits were small and less abundant. Microscopic examination of kidney sections derived from EG induced urolithiatic rats showed calcification inside the tubules which causes dilation of the proximal tubules. Co-treatment with Megarajanga Chooranam decreased the calcification in different parts of therenal tubules and also prevented damages to the tubules and calyxes. Organ-body weight ratio is amarker of cell constriction and inflammation. The non-significant effect on the rat kidney-bodyweight ratio following the administration of various doses of the Megarajanga Chooranam suggests that the drug did not induce inflammation or constriction of the kidney cells.

Pathologic studies have shown that the renal failure from EG is associated with proximal tubule cell necrosis leading to production of several metabolites (glycol aldehyde, glycolate, glyoxylate and oxalate, in that order) and accumulation of large calcium oxalate monohydrate crystals in tubular lumen.

An Ayurvedic compound preparation (Cystone) was found to contain water solublesubstances, which inhibited the initial precipitationof calcium and phosphate ions in the form of a mineral phase bound to the organic matrix and the subsequent growth of the preformed mineral phase.In the present study, concurrent administration of EG with cystone / Megarajanga Chooranam causes significant curative effect in EG induced changes. The effect is dose dependent. The effectiveness of Megarajanga Chooranam is comparable to cystone.

Renal Stone denotes stones originating anywhere in the urinary tract, including the kidneys, ureters and bladder. The formation of stones in the urinary tract affects 5–10% of the population in Europe and North America. In most populations the occurrence of urolithiasis in men is two to three times higher than in women as testosterone enhances whereas estrogen inhibits calcium oxalate stone formation. The formation of calcium oxalate stone is a multi-step process and includes–nucleation, crystal growth, crystal aggregation and crystal retention which further result in precipitation of certain substances within urine. This process is favored in the presence of a supersaturated milieu which is necessary for precipitating crystal. Thus supersaturation acts as a driving force for stone formation. Additionally another theory of stone formation was identified and concluded as an imbalance between promoter (calcium, oxalate, uric acid, inorganic phosphate etc) and inhibitors (citrates, magnesium, potassium, pyrophosphate and urinary glycoprotein etc).

Furthermore, reactive oxygen species or free radicals (species with one or more unpaired electrons) generated due to oxidative stress, damages epithelium of kidney or bladder, thereby producing a favorable environment for crystal attachment to surface. As a result of these, the stone may not be able to travel through the ureter, causing pain and possibly an obstruction, blocking the flow of urine out of the kidney. Severe pain or aching in the back on one or both sides, sudden spasms of excruciating pain, bloody, cloudy or smelly urine,

feeling of being sick, a frequent urge to urinate, or a burning sensation during urination, fever and chills, etc are commonly observed symptoms in the patients. Patients suffering from diseases like hyperparathyroidism, renal tubular acidosis, cystinuria, hypercalciuria, hyperoxaluria, crohn's disease etc. are more prone to stone formation.

The renal tissue of EG along with Megarajanga Chooranam shows only few stray areas of calcification in glomeruli and normal tubular structures with no congestion in blood vessels. The renal tissue of standard drug treatment still shows moderate calcification in many tubules and few glomeruli. It has been reported that the kidneys are the principle target organfor ethylene glycol toxicity and administration of ethylene glycol for 3 weeks resulted in insignificant urinary oxalate excretion and deposition of crystals in kidney, hence in our study ethylene glycol was chosen to induce lithiasis. Following the induction of lithiasis the urinary volume and composition were found to be altered.

In our study also the urinary output was markedly decreased in lithiatic control rats on day 28, however in Megarajanga Chooranam and standard treated rats the urinary volumes were increased when compared to that lithiatic Group. This suggested that Megarajanga Chooranam might have moderate diuretic effect. Following ethylene glycol administration the excretion of calcium, oxalate, phosphate and protein were found to be increased in lithiatic group while in standard, test groups these levels were significantly decreased ($P<0.01$).

On contrary to this the magnesium level was decreased in lithiatic group while in standard and Megarajanga Chooranam treated groups those levels were increased significantly ($P<0.01$). The serum creatinine levels of Megarajanga Chooranam treated rats restored to normal limits and the creatinine clearance was also found to be improved. The findings of the histopathological studies suggested that no microcrystalline deposition and kidney damage in the

Megarajanga Chooranam treated groups. All these observations enabled us to confirm the inhibitory potential of Megarajanga Chooranam on ethylene glycol induced lithiasis.

CONCLUSION

The presented data indicate that administration of the Megarajanga Chooranam to rats with ethylene glycol induced lithiasis reduced the formation of urinary stones, supporting clinical information regarding antiurolithiatic activity of the Megarajanga Chooranam. The mechanism underlying this effect is still unknown, but is apparently related to diuresis and lowering of urinary concentrations of stone forming constituents. These effects could conclude the antiurolithiatic property of Megarajanga Chooranam.

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Table 1: Diuretic activity of MegarajangaChooranam in rats

Group	Treatment and Dose	Volume of Urine (ml/4hrs)	Sodium (mMol/l)	Potassium (mMol/l)	Chloride (mMol/l)
I	Saline (10ml/ kg)	0.82±0.15	74.7±5.3	65.4±4.2	91.2±6.4
II	MRC (250 mg/ kg)	0.78±0.12^{b}	88.0±6.9^{b}	78.6±6.1^{c}	110.8±10.1^{c}
III	MRC (500 mg/ kg)	1.12±0.15^{b}	100.3±6.4^{c,*}	89.2±5.8*	122.0±6.2*
IV	Frusemide (20 mg/ kg)	4.10±0.22**	125.1±4.8**	102.1±6.5**	142.7±8.8**

All values are expressed as mean ±S.E.M for six rats in each group.

Comparisons made between ***p<0.001; **p<0.01;*p<0.05 V_s control; ^{a}p<0.001; ^{b}p<0.01; ^{c}p<0.05 V_s Standard.

Table 2: Estimation of Urinary Electrolytes of Normal and Urolithiatic Rats.

S.No	Group & Drug Treatment	Estimation of Urinary Electrolytes		
		Oxalate(mg/dl)	Calcium(mg/dl)	Phosphate(mg/dl)
1	Normal control (Saline)	0.35±0.05	2.66±0.12	3.21±0.03
2	Calculi induced(0.75% EG)	2.28±0.05$^{©,x}$	8.17±0.36$^{©,x}$	9.12±0.17$^{©,x}$
3	Standard (Cystone 750 mg/kg)	1.22±0.05^{x}	3.19±0.20^{x}	3.67±0.08^{x}
4	T_1 (MRC250 mg/kg)	1.25±0.20***	5.84±0.13^{a,***}	6.20±0.06^{a,***}
5	T_2 (MRC500 mg/kg)	0.76±0.12***	4.33±0.12^{b,***}	4.98±0.13^{a,***}

All values are expressed as mean ±S.E.M for six rats in each group.
Comparisons made between
^{a}p<0.001,^{b}p<0.01,^{c}p,<0.05; T_1,$T_2$$V_s$Standard.
***p<0.001,**p<0.01,*p<0.05 ; T_1,$T_2$$V_s$ Calculi induced.
$^{©}$p<0.001,$^{\alpha}$p<0.01,@p<0.05; Calculi induced $V_s$normal control.
^{x}p<0.001,^{y}p<0.01,^{z}p,<0.05; Calculi induced V_sStandard., One-way ANOVA followed by Tukey test.

Table 3: Estimation of Kidney Homogenate Electrolytes of Normal And Urolithiatic Rats.

S.No	Group &Drug Treatment	Estimation of Kidney Homogenate Parameters		
		Oxalate(mg/dl)	Calcium(mg/dl)	Phosphate(mg/dl)
1	Normal (Saline)	0.172±0.07	3.668±0.44	2.46±0.07
2	Positive control (0.75% EG)	1.742±0.09$^{©,x}$	6.12±0.22$^{©,x}$	4.36±0.16$^{©,x}$
3	Standard (Cystone 750 mg/kg)	0.569±0.06^{x}	4.15±0.15^{x}	3.05±0.10^{x}
4	T_1 (MRC250 mg/kg)	1.156±0.09a,***	5.30±0.22^{c}	2.96±0.17***
5	T_2 (MRC500 mg/kg)	0.768±0.08***	4.19±0.16***	2.67±0.08***

All values are expressed as mean ±S.E.M for six rats in each group.
Comparisons made between
$^{a}p<0.001, ^{b}p<0.01, ^{c}p,<0.05$; $T_1, T_2 V_s$ Standard.
$^{***}p<0.001, ^{**}p<0.01, ^{*}p<0.05$; $T_1, T_2 V_s$ Calculi induced.
$^{©}p<0.001, ^{\alpha}p<0.01, @p<0.05$; Calculi induced $V_s$normal control.
$^{x}p<0.001, ^{y}p<0.01, ^{z}p,<0.05$; Calculi induced V_sStandard., One-way ANOVA followed by Tukey test.

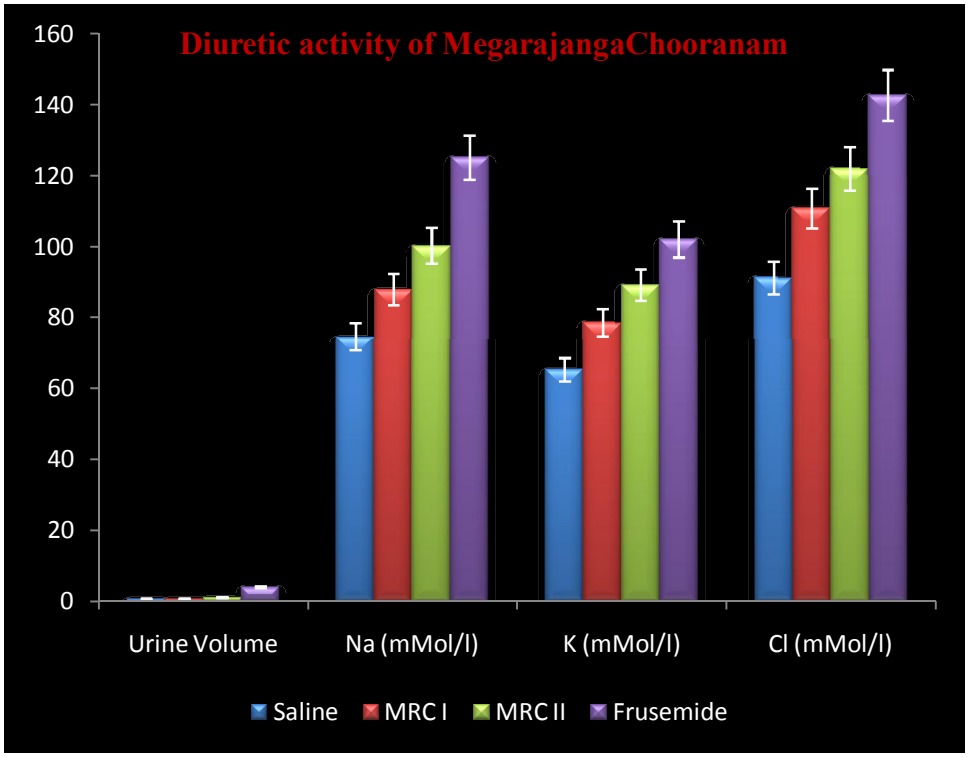
Table 4: Estimation of Serum Parameters of Normal and Urolithiatic Rats.

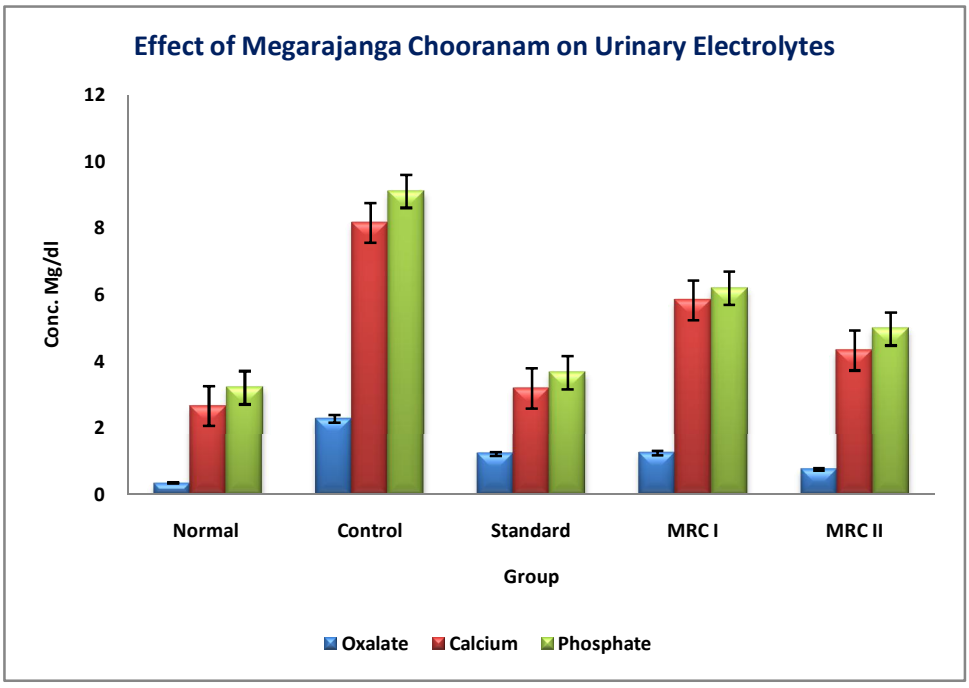
S.No	Group & Drug Treatment	Estimation of Serum Parameters		
		BUN (mg/dl)	Creatinine (mg/dl)	Uric acid (mg/dl)
1	Normal (Saline)	20.31±0.28	0.702±0.06	4.86±0.07
2	Positive control (0.75% EG)	28.66±0.41$^{©,x}$	0.924±0.07	6.93±0.10$^{©,x}$
3	Standard (Cystone 750 mg/kg)	23.91±0.32^{x}	0.846±0.09^{x}	5.26±0.08^{x}
4	T_1 (MRC250 mg/kg)	26.81±0.52^{a,*}	0.899±0.10	6.33±0.09^{a,***}
5	T_2 (MRC500 mg/kg)	24.35±0.40***	0.828±0.12	6.00±0.11^{a,***}

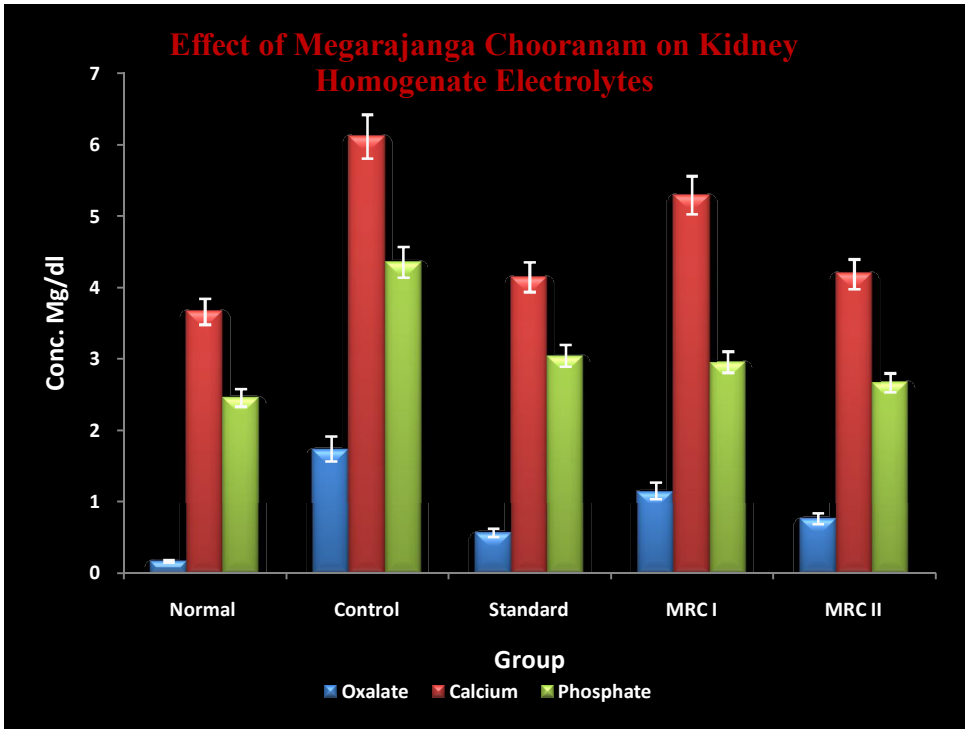
All values are expressed as mean ±S.E.M for six rats in each group.

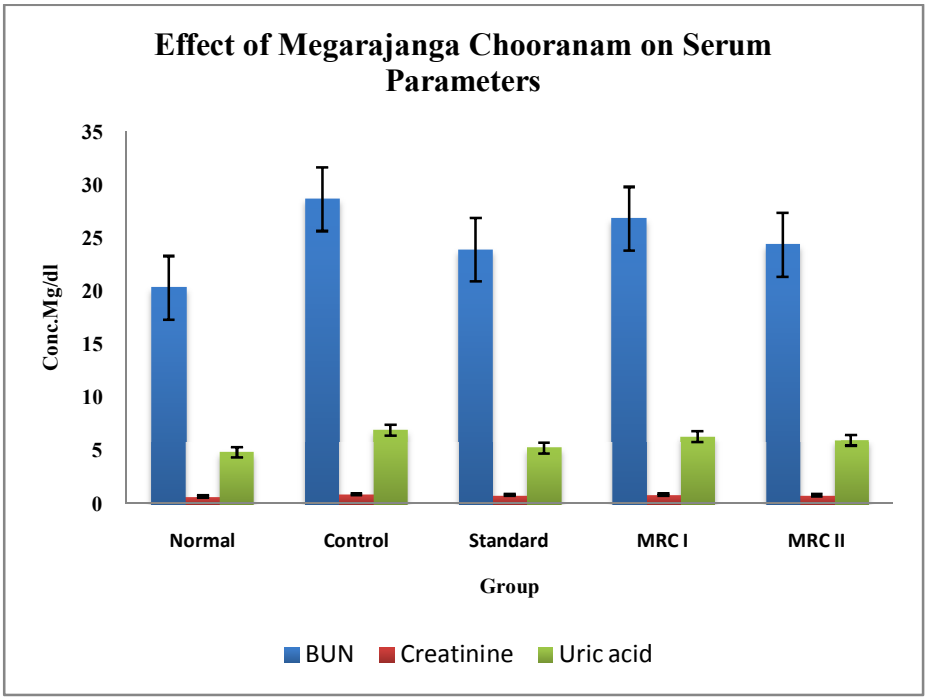
Comparisons made between

$^{a}p<0.001, ^{b}p<0.01, ^{c}p,<0.05$; $T_1, T_2 V_s$ Standard.
$^{***}p<0.001, ^{**}p<0.01, ^{*}p<0.05$; $T_1, T_2 V_s$ Calculi induced.
$^{©}p<0.001, ^{\alpha}p<0.01, @p<0.05$; Calculi induced $V_s$normal control.
$^{x}p<0.001, ^{y}p<0.01, ^{z}p,<0.05$; Calculi induced V_sStandard., One-way ANOVA followed by Tukey test.

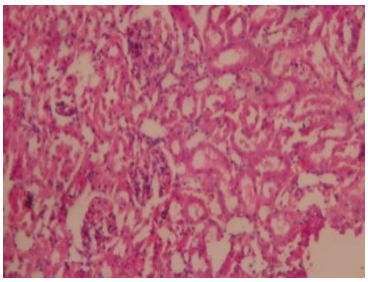

Diuretic activity of MegarajangaChooranam
160
140
120
100
80
60
40
20
0
Urine Volume
Na (mMol/l)
K (mMol/l)
Cl (mMol/l)
Saline
MRC I
MRC II
Frusemide


Effect of Megarajanga Chooranam on Urinary Electrolytes
Conc. Mg/dl
12
10
8
6
4
2
0
Normal
Control
Standard
MRC I
MRC II
Group
Oxalate
Calcium
Phosphate

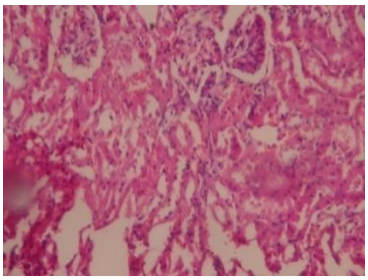

Effect of Megarajanga Chooranam on Kidney Homogenate Electrolytes
Conc. Mg/dl
0
1
2
3
4
5
6
7
Normal
Control
Standard
MRC I
MRC II
Group
Oxalate
Calcium
Phosphate


Effect of Megarajanga Chooranam on Serum Parameters
Conc.Mg/dl
0
5
10
15
20
25
30
35
Normal
Control
Standard
MRC I
MRC II
Group
BUN
Creatinine
Uric acid

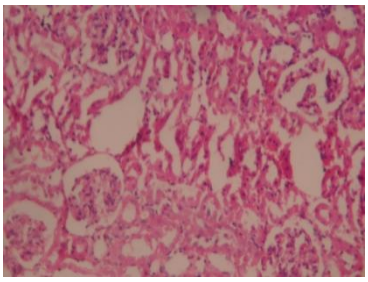
HISTOPATHOLOGICAL STUDY OF TRIAL DRUGS



Lithiatic Control 1



Lithiatic Control



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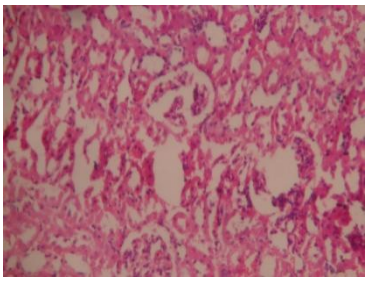

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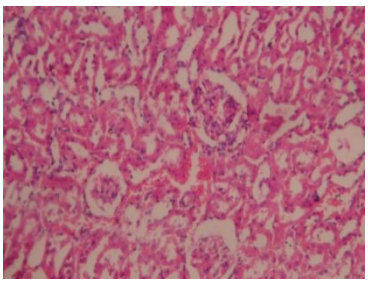
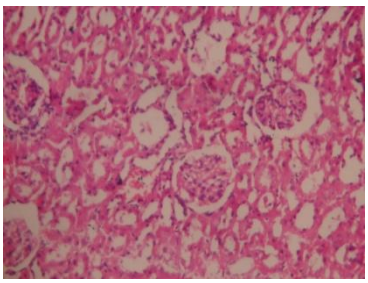
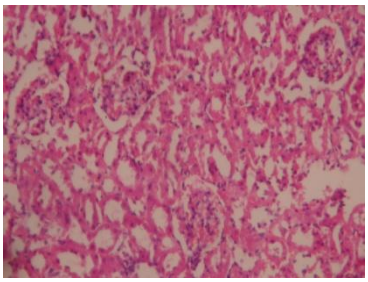


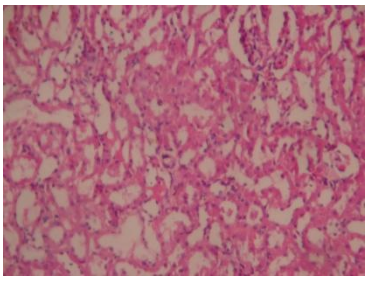
MRC 500 mg 1



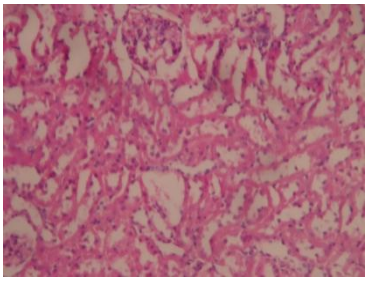
MRC 500 mg



Normal



Standard 1



Standard

ANNEXURE - 3

ACUTE AND SUB ACUTE TOXICITY STUDY ON MEGARAJANGA CHOORANAM

Animals:

Mice of either sex weighing 25-30g and rats weighing 210-240g were obtained from the animal house of Vels University. The animals were used with the approval of the Institute animal ethics committee and obtained from Vels University, Chennai. They were fed with a balanced standard pellet diet and maintained under standard laboratory conditions, providing 24-28^{0}C temperature, standard light cycle (12 h light, 12 h dark) and water ad libitum. Animals were kept in cages with raised floors of wide mesh to prevent coprophagy. Animal welfare guidelines were observed during the maintenance period and experimentation. The rats were randomly assigned to control and different treatment groups, six animals per group. The animals were acclimatized for one week under laboratory conditions.

ACUTE TOXICITY STUDY-OECD 425 GUIDELINES

Acute oral toxicity test for the Megarajanga Chooranam was carried out as per OECD Guidelines 425. As with other sequential test designs, care was taken to ensure that animals are available in the appropriate size and age range for the entire study. The test substance is administered in a single dose by gavage using a stomach tube or a suitable intubation cannula. The fasted body weight of each animal is determined and the dose is calculated according to the body weight. After the substance has been administered, food was withheld for a further 2 hours in mice. The animals were observed continuously for the first 4 h and then each hour for the next 24 h and at 6 hourly intervals for the following 48 h after administering of the test drug, to observe any death or changes in general behaviour and other physiological activities. Single animals are dosed in sequence usually at 48 h intervals. However, the time interval between dosing is determined by the onset, duration, and severity of toxic signs. Treatment of an animal at the next dose was delayed until one is confident of survival of the previously dosed animal.

Observation of toxicity signs: General behavior, respiratory pattern, cardiovascular signs, motor activities, reflexes, change in skin and fur, mortality and the body weight changes were monitored daily. The time of onset, intensity, and duration of these signs, if any, was recorded.

SUB-ACUTE TOXICITY

In a 28-days sub acute toxicity study, twenty four either sex (3+3) rats were divided into four groups of 6 rats each. Group I that served as normal control was administered with distilled water (p.o.) while groups II, III and IV were administered daily with the Megarajanga Chooranam (p.o.) for 28 days at a dose of 100, 250 and 500mg/kg respectively. The animals were then observed daily for gross behavioural changes and any other signs of subacute toxicity. The weight of each rat was recorded on day 0 and weekly throughout the course of the study, food and water consumption per rat was calculated. At the end of the 28 days they were fasted overnight, each animal was anaesthetized with diethylether, following which they were then dissected and blood samples were obtained by cardiac puncture into heparinised tubes. The blood sample collected from each rat was centrifuged with 3000 X g at 4^{o}C for 10 min to separate the serum and used for the biochemical assays.

Hematological and blood biochemical analyses:

At the end of the study, all animals were kept fasted for 16-18 h and then anesthetized with anesthetic ether on the 28th day. Blood samples for hematological and blood chemical analyses were taken from retro orbital vein. Heparinized blood samples were taken for determining complete blood count (white blood cell count, differential white blood cell count, platelet count, red blood cell count, hematocrit, and hemoglobin) by semiautomated hematology analyzer. The serum from non-heparinized blood was carefully collected for blood chemistry and enzyme analysis creatinine, total protein, albumin, total and direct bilirubins, serum glutamate-oxaloacetate transaminase (SGOT), serum glutamate pyruvate transaminase (SGPT), and alkaline phosphatase (ALP)) were automatically determined using autoanalyzer.

Necropsy:

All rats were sacrificed after the blood collection. The positions, shapes, sizes and colors of internal organs were evaluated. The Spleen, Testes, Pancrea, Lung, Liver, Brain, Heart, Stomach, Intestine, Bone, Ovary, and Kidney tissues were excised from all rats to visually detect gross lesions, and weighed to determine relative organs' weights and preserved in 10% neutral formalin for histopathological assessment. The tissues were embedded in paraffin, and then sectioned, stained with haematoxylin and eosin and were examined microscopically.

Statistical analysis

Values were represented as mean ± SEM. Data were analysed using one-way analysis of variance (ANOVA) and group means were compared using the Tukey-Kramer Multiple Comparison Test using GraphPad Instat-V3 software. P values < 0.05 were considered significant.

RESULTS

Animals were not shown any significant toxic clinical signs during the dosing period of 28 days. All animals from control and all the treated dose groups survived throughout the dosing period of 28 days. Results of body weight determination of animals of control and different dose groups exhibited comparable body weight gain throughout the dosing period of 28 days. During dosing period, the quantity of food consumed by animals from different dose groups was found to be comparable and normal with that of control animals. Ophthalmoscopic examination of animals in control and test product– treated groups did not reveal any major and remarkable abnormality.

These tests conducted on the experimental animals at termination and recorded did not reveal any abnormalities. Urine analysis data of control group and treated group of animals determined in week 4 did not reveal any abnormalities. Mean Relative Organ Weights are found to be comparable and normal. Gross pathological examination of animals in control as well as the treated groups did not reveal any abnormalities. The

results of haematological investigations revealed no major changes in the values of different parameters investigated when compared with control; However, the increase or decrease in the values obtained was within normal biological and laboratory limits. Results of Biochemical investigations revealed normal.

CONCLUSION

In the present study, no toxic effect was observed upto 500mg/kg of Megarajanga Chooranam treated via oral route over a period of 28 days. So, it can be concluded that the Megarajanga Chooranam can be prescribed for therapeutic use in human with the dosage recommendations of upto 500mg/kg. body weight p.o.

Table 1: Dose finding experiment and its behavioral Signs of Toxicity

No	Dose mg/ kg	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1.	**2000**	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-
2	**5000**	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-

1. Alertness 2. Aggressiveness 3. Pile erection 4. Grooming 5. Gripping 6. Touch Response 7. Decreased Motor Activity 8. Tremors 9. Convulsions 10. Muscle Spasm 11. Catatonia 12. Muscle relaxant 13. Hypnosis 14. Analgesia 15.Lacrimation 16. Exophthalmos 17. Diarrhoea 18. Writhing 19. Respiration 20. Mortality

Table 2. Body wt (g) of albino rats exposed to Megarajanga Chooranam for 28days

Dose (mg/kg/ day)	Days				
	1	**7**	**14**	**21**	**28**
Control	123.45±8.05	125.56±6.00	124.25±5.00	128.05±5.14	132.62±5.15
100	127.10±4.31	129.21±5.15	132.12±5.62	135.64±6.00	136.45±4.00
250	124.26±6.44	128.13±6.88	131.15±5.10	134.18±5.15	137.30±6.11
500	122.13±8.27	125.62±5.34	128.12±5.82	131.02±6.30	134.62±5.35

Values are mean of 6 animals ± S.E.M. (Dunnet's test). $^{ns}P>0.05$..

Table 3. Food (g/day) intake of rats exposed to Megarajanga Chooranam for 28days

Dose (mg/kg/ day)	Days (gms/rats)				
	1	7	14	21	28
Control	44.13±2.82	44.65±2.15	45.42±2.30	45.46±2.42	48.48±3.00
100	42.20±2.12	45.13±2.46	46.58±2.26	47.28±2.52	48.12±3.02
250	40.32±2.10	44.19±2.59	45.68±2.52	45.62±3.18	46.10±3.00
500	43.12±2.48	45.24±2.83	46.11±2.72	45.18±2.88	45.56±3.12

Values are mean of 6 animals ± S.E.M. (Dunnet's test). $^{ns}P>0.05$.

Table 4. Water (ml/day) intake of rats exposed to Megarajanga Chooranam for 28days

Dose (mg/kg/day)	Days(ml/rat)				
	1	7	14	21	28
Control	55.24±2.80	55.00±3.26	52.10±3.15	52.30±3.18	52.28±3.21
100	55.19±2.42	55.20±3.41	45.20±4.02	46.10±3.00	40.50±2.82**
250	48.72±2.81	48.12±3.77	42.72±3.34	44.15±2.91	45.12±3.22
500	50.23±2.56	56.16±3.10	54.25±3.18	49.02±3.19	45.00±3.16

Values are mean of 6 animals ± S.E.M. (Dunnet's test). **P<0.01.

Table 5. Effect of treatment with Mega Rajanga Chooranam biochemical parameters

Dose (mg/kg)	Control	100 mg/kg	250 mg/kg	500 mg/kg
Total Bilirubin (mg/dL)	0.211±0.05	0.210±0.06	0.214±0.05	0.210±0.04
Bilirubin direct (mg/dL)	0.1±0.04	0.1±0.05	0.1±0.04	0.1±0.05
ALP (U/L)	71.05±2.5	70.11±2.8	69.21±3.2	71.30±2.8
SGOT (U/L)	73.10±2.6	74.12±2.5	72.10±3.0	75.22 ± 2.1
SGPT(U/L)	81.4±3.2	82.10±3.0	82.12±2.7	80.21±2.2
Total Protein(g/dl)	10.10±1.02	10.14±0.80	9.58±0.77	9.51±0.96
Albumin(g/dl)	3.27±0.25	3.51±0.24	3.44±0.13	3.22±0.12
Globulin(g/dl)	6.00±0.18	5.48±0.20	4.90±0.24*	4.62±0.31**
Urea (mg/dL)	55.25±1.26	54.41±3.25	55.0±2.14	54.18±1.32
Creatinine (mg/dL)	28.22±3.0	31.17±3.2	29.42±3.12	29.11 ± 3.0
Uric acid (mg/dL)	1.6±0.10	1.6±0.12	1.6±0.16	1.6±0.10
Na m.mol	142.10±4.22	141.0±3.15	142.10±4.02	141.10±3.46
K m.mol	20.18±2.12	19.75±2.44	20.28±2.20	20.25±2.31
Cl m.mol	100.52±4.18	101.00±5.54	100.99±4.00	102.16±4.08

Values are mean ± S.E.M. (Dunnet 't' test). *P<0.05; **P<0.01 Vs control..

Table 6. Hematological parameters after 28days treatment with Megarajanga Chooranam

Parameter	Control	100 mg/kg	250 mg/kg	500 mg/kg
RBC (mm^3)	8.10±0.45	7.55±0.44	8.14±0.46	8.40±0.54
HB (%)	14.24±0.36	14.45±0.38	14.51±0.36	14.15±0.32
Leukocyte ($x10^6$/mL)	11.14±1.27	10.22±1.25	10.82±1.26	10.74±1.35
Platelets ($X10^5$/µl)	1.25±0.12	1.32±0.14	1.35±0.14	1.38±0.14
MCV (g/l)	85.62±4.5	85.24±5.30	86.12±4.22	84.54±5.24
Neutrophil (%)	54.22±3.18	52.12 ±3.22	50.46±3.0	51.22±3.6
Lymphocytes (%)	44.12±1.28	45.10±3.2	45.65±3.2	45.11±3.2
Eosinophil's (%)	5.0±0.4	5.0±0.4	5±0.3	5±0.4
Monocytes (%)	3.0±0.02	3.0±0.3	3.0±0.3	3.0±0.3
Basophils (%)	0±0	0±0	0±0	0±0
ESR(mm)	1±00	1±00	1±00	1±00
PCV	45.12±3.28	44.16±3.12	43.56±3.22	42.12±3.24

Values are mean ± S.E.M. (Dunnet 't' test). $^{ns}P>0.05$; Vs Control N=6.

Table-8. Lipid Profile

Dose (mg/kg)	Control	100 mg/kg	250 mg/kg	500 mg/kg
Total cholesterol (mg/dL)	40.20±2.45	41.10±2.26	42.14±3.45	42.10±3.18
HDL(mg/dL)	14.28±2.30	14.20±1.72	14.00±1.55	14.29±2.21
LDL(mg/dL)	43.12±2.45	44.41±3.10	43.62±3.39	43.21±3.24
VLDL(mg/dl)	16.61±2.70	15.84±2.13	16.20±1.58	15.45±1.20
Triglycerides (mg/dl)	85.80±3.02	85.32±2.78	86.41±3.04	85.34±2.26
Blood glucose(mg/dl)	126.21±3.84	126.56±3.12	127.00±4.13	125.44±2.88

Values are mean ± S.E.M. (Dunnet 't' test). $^{ns}P>0.05$; Vs Control N=6.

Table-9 Urine Analysis

Parameters	Control	100 mg/kg	250 mg/kg	500 mg/kg
Colour	Yellow	Yellow	Yellow	Yellow
Transparency	Clear	Slightly turbid	Slightly cloudy	Slightly turbid
Specific gravity	1.010	1.010	1.010	1.010
PH	>7.2	>8.0	>8.0	>9.0
Protein	Nil	3+	3+	3+
Glucose	Nil	Nil	Nil	Nil
Bilirubin	-ve	-ve	-ve	-ve
Ketones	-ve	+ve	+ve	+ve
Blood	Absent	Absent	Absent	Absent
Urobilinogen	Normal	Abnormal	Abnormal	Abnormal
Pus cells	0-cells/HPF	1-cell/HPF	2-cells/HPF	1-cell/HPF
RBCs	Nil	Nil	0-1cells/HPF	Nil
Epithelial cells	Nil	1-cell/HPF	Nil	1-cell/HPF
Crystals	Nil	Nil	Nil	Nil
Casts	Nil	Nil	Nil	Nil
Others	Bacteria seen	Bacteria seen	Bacteria seen	Bacteria seen

Table 10. Effect of oral administration of a Megarajanga

Chooranam on organ weight

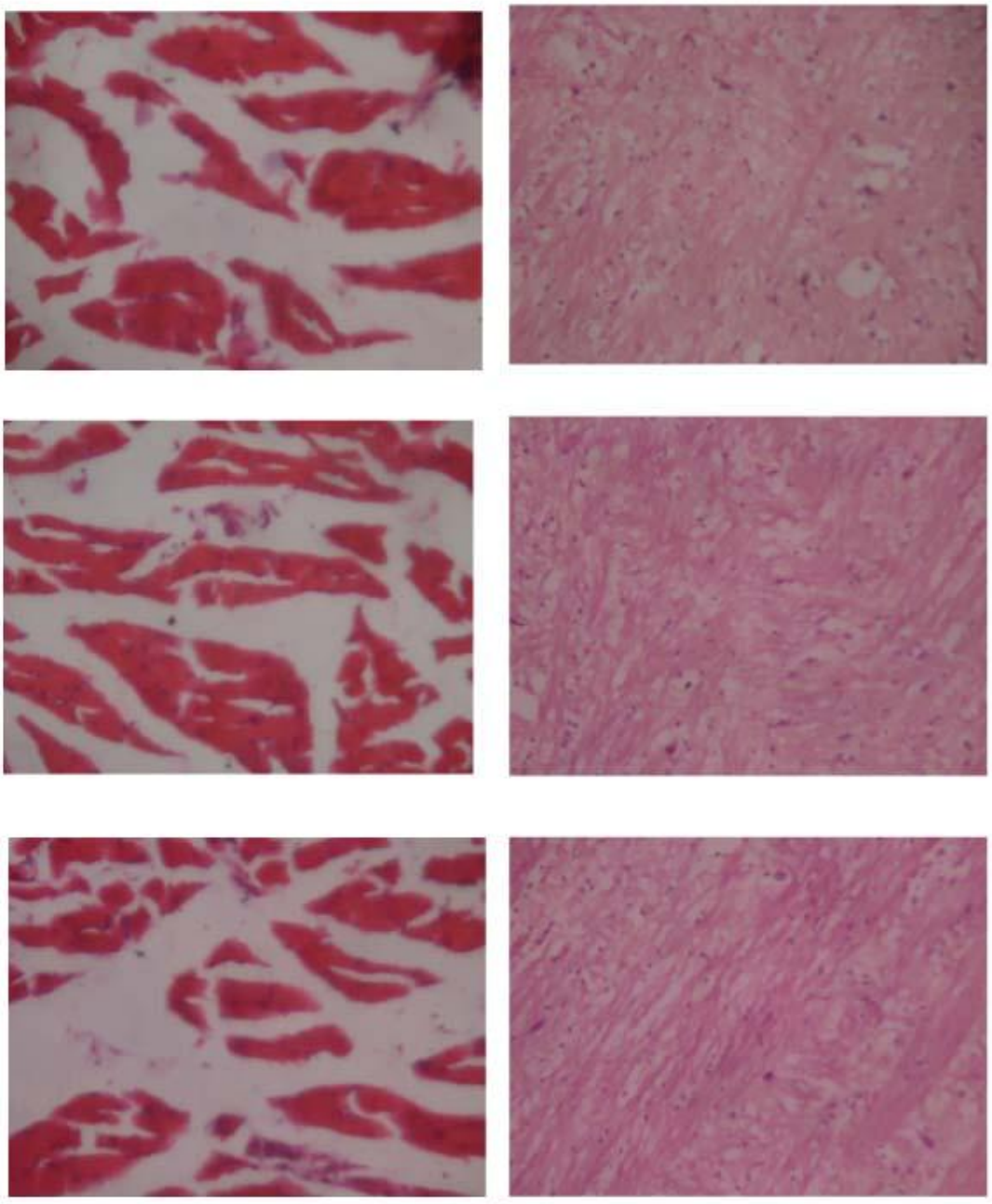
Dose (mg/kg)	Control	100 mg/kg	250 mg/kg	500 mg/kg
Liver (g)	5.22±0.15	5.30±0.15	4.72±0.14	5.12±0.12
Heart (g)	0.60±0.04	0.61±0.05	0.65±0.04	0.57±0.05
Lung (g)	1.48±0.06	1.42±0.14	1.46±0.26	1.50±0.12
Spleen (g)	0.69±0.06	0.69±0.04	0.69±0.05	0.65±0.04
Ovary (g)	1.68±0.15	1.79±0.15	1.76±0.16	1.74±0.15
Testes (g)	1.45±0.12	1.48±0.12	1.45±0.14	1.45±0.12
Brain (g)	1.52±0.16	1.57±0.13	1.55±0.12	1.50±0.15
Kidney (g)	0.72±0.04	0.70±0.04	0.72±0.05	0.71±0.04
Stomach (g)	1.38±0.14	1.36±0.10	1.37±0.12	1.36±0.10

Values are mean of 6 animals ± S.E.M. (Dunnet's test). $^{ns}P>0.05$; Vs Control N=6.

HISTOPATHOLOGY

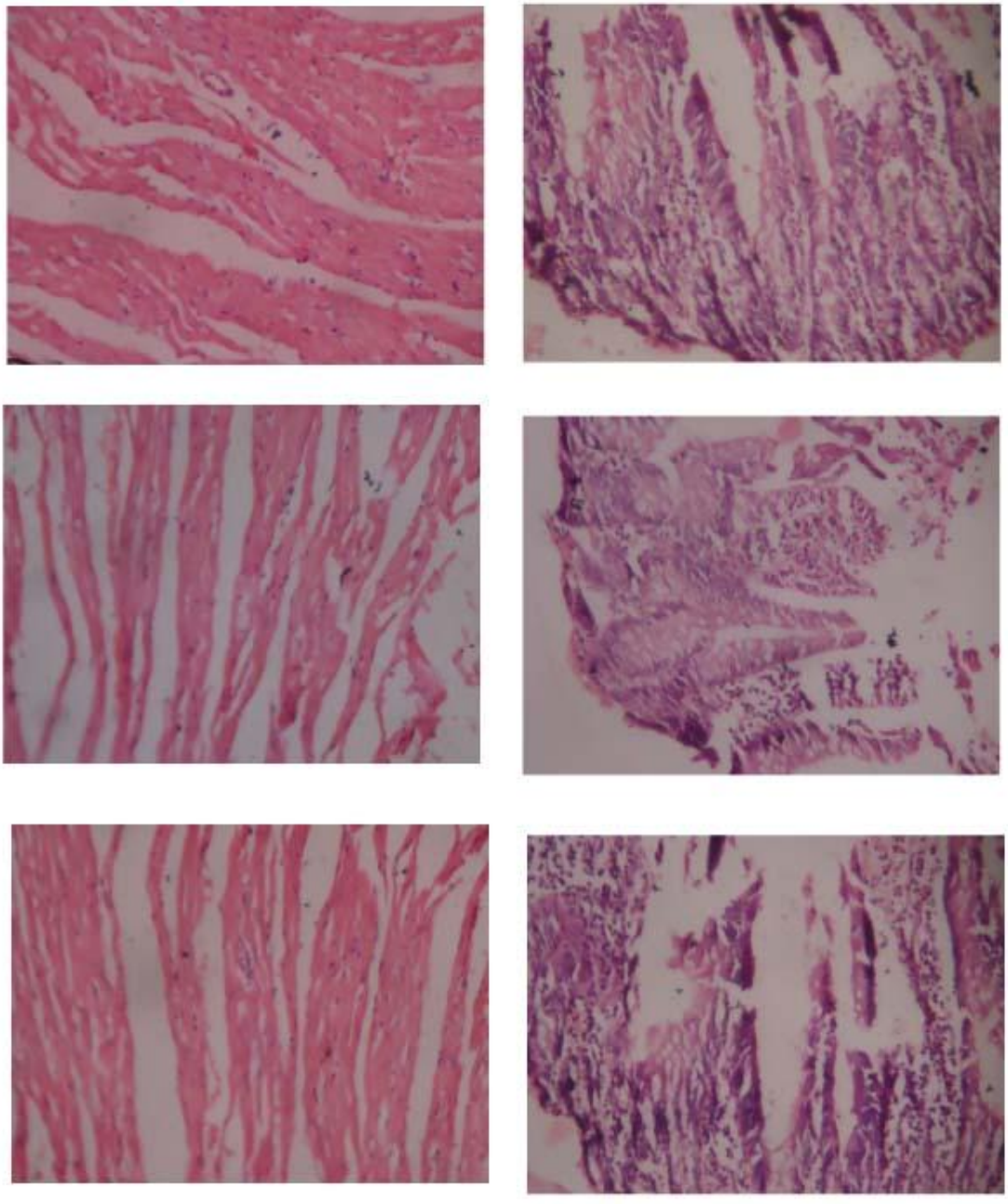
Bone

Brain



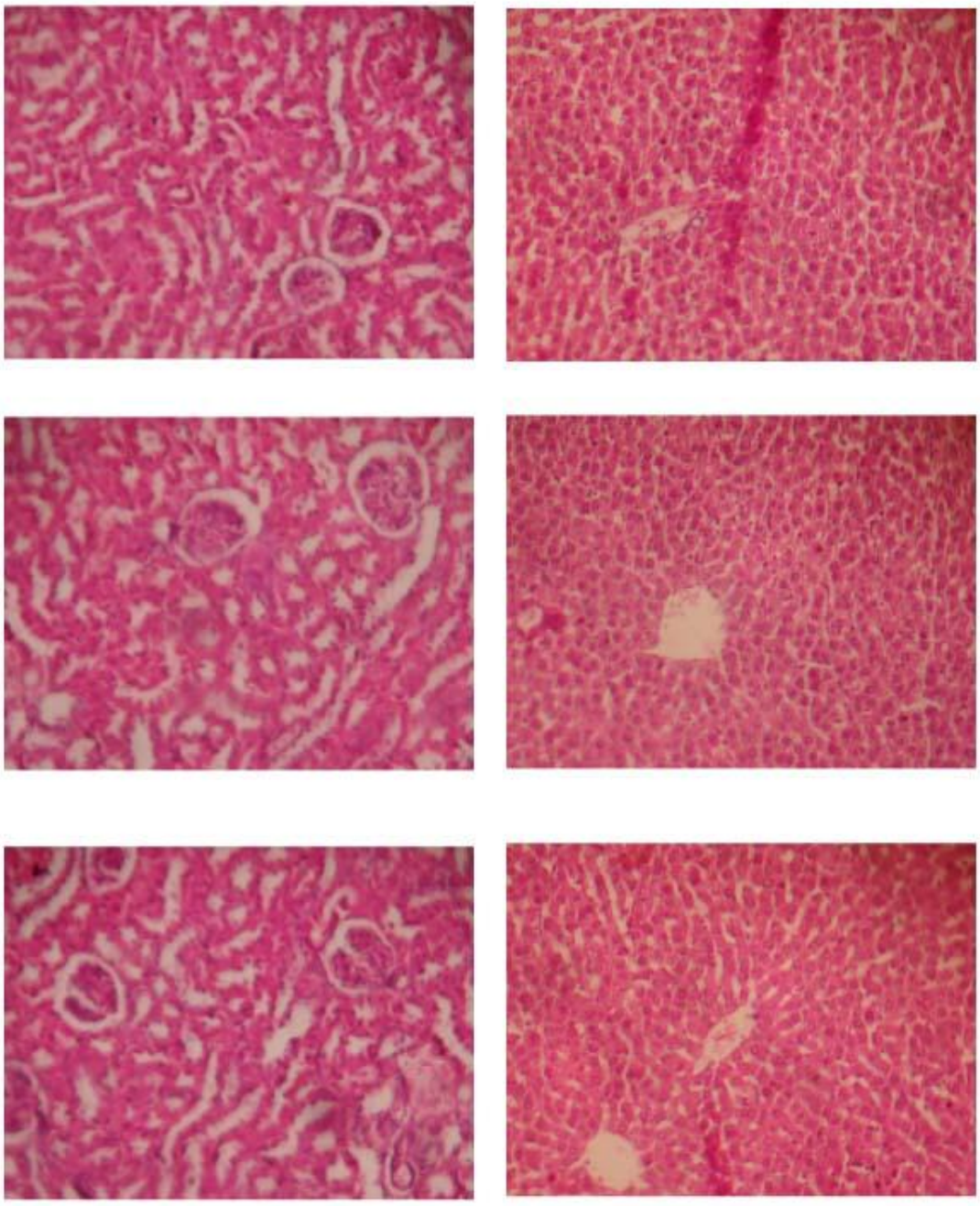
Heart

Intestine



Kidney

Liver



ANNEXURE - 4

STATISTICAL ANALYSIS OF CLINICAL STUDIES

All collected data were entered into ms excel software using different columns as variables and rows as patients. The probability value 0.05 was taken and as statistically significant level. Before and after treatment data was analyzed using paired 't' test to determine the significance of treatment. The quantity variables were represented by means of mean ± standard deviation and qualitative variables by means of percentage. Basic descriptive statistics include frequency distributions and cross tabulations.

NO. OF CALCULI-RIGHT KIDNEY

BT	Mean	S.D.	T.Value	Pvalue
	1.516	± 0.889	5.4292	0.0000
AT	0.677	± 0.747		

The average ± standard deviation for Rt. Kidney calculi before and after treatment, were 1.5 ± 0.889 & 0.6 ± 0.747 respectively which is statistically highly significant (P<0.0001)

NO. OF CALCULI-LEFT KIDNEY

BT	Mean	S.D.	T.Value	Pvalue
	1.548	± 0.994	4.2515	0.0001
AT	0.709	± 0.782		

The average ± standard deviation for Lt. Kidney calculi before and after treatment, were 1.54 ± 0.994 & 0.70 ± 0.782 respectively which is statistically highly significant (P<0.0001)

CALCULI MEASUREMENT - RIGHT KIDNEY

BT	Mean	S.D.	T.Value	Pvalue
	6.783	± 4.358	6.912	0.0000
AT	2.5	± 2.883		

The average ± standard deviation for Rt. Kidney calculi measurement before and after treatment, were 6.7 ± 4.358 & 2.5 ± 2.883 respectively which is statistically highly significant (P<0.0001)

CALCULI MEASUREMENT - LEFT KIDNEY

BT	Mean	S.D.	T.Value	Pvalue
	7.10	± 5.106	6.1425	0.0000
AT	0.90	± 3.512		

The average ± standard deviation for Lt. Kidney calculi measurement before and after treatment, were 7.1 ± 5.106 & 2.9 ± 3.512 respectively which is statistically highly significant (P<0.0001)

CLINICAL MANIFESTATIONS

BT	Mean	S.D.	T.Value	Pvalue
	2.95	± 1.108	12.83	0.0000
AT	0.57	± 0.675		

The average ± standard deviation for clinical manifestations before and after treatment, were 2.9 ± 1.108 & 0.5 ± 0.675 respectively which is statistically highly significant (P<0.0001)

ANNEXURE - 5

CONSENT FORM

I certify that I have disclosed all the details about the study in the terms readily understood by the patient.

DATE: SIGNATURE
NAME:

CONSENT BY THE PATIENT

I have been informed to my satisfaction by the attending physician for the purpose of the clinical trial and the nature of the drug treatment and follow up including the lab investigation to be performed to monitor and safeguard my body functions.

I am aware of my right to opt out of the trial at any time during the course of the trial without having to give reasons for doing so.

I, exercising my free power of choice, hereby give my consent to be included as a subject in the clinical trial of **MEGARAJANGA CHOORANAM** for the treatment of **KALLADAIPPU NOI.**

DATE:

SIGNATURE NAME

நோயாளியின் ஒப்புதல் படிவம்

திரு._____ஆகிய நான் _____வயது,
(_____
_____
வசிக்கும் இடம்.) என் சுய நினைவுடன் எழுதிக் கொடுக்கும் ஒப்புதல் படிவம்.

நான் **கல்லடைப்பு** என்னும் நோயால் பாதிக்கப்பட்டு சென்னை, அரசு சித்த மருத்துவ கல்லூரியில் (இடம்: அறிஞர் அண்ணா இந்திய மருத்துவமனை, அரும்பாக்கம், சென்னை-106.) நடத்தப்படும் சித்த மருத்துவ ஆராய்ச்சி மூலம் சிகிச்சை பெற என் சுய நினைவுடன் முழுசம்மதத்தையும் தெரிவித்துக்கொள்கிறேன்.

இந்த ஆராய்ச்சியின் நோக்கம், மருத்துவம் செய்யும் முறை, தொடர்கண்காணிப்பு மற்றும் என் உடல் நலம் குறித்த மருத்துவ பரிசோதனைகளைப் பற்றிய விரிவான விளக்கம் எனக்கு மருத்துவம் செய்யும் மருத்துவர் மூலம் தெளிவுபடுத்தப்பட்டுள்ளது. இந்த ஆராய்ச்சியில் பங்குகொள்ளும் என் சம்மதத்திற்கு யாருடைய நிர்பந்தமும் காரணமில்லைஎன்பதை தெரிவித்துக்கொள்கிறேன்.

இப்படிக்கு,

பெயர் :
முகவரி :

நாள் :

ANNEXURE - 6

OP/IP CASE SHEET PROFORMA

POST GRADUATE DEPARTMENT, POTHU MARUTHUVAM

(BRANCH-I)

GOVT. SIDDHA MEDICAL COLLEGE & HOSPITAL, CHENNAI-106

PROFORMA FOR 'KALLADAIPU'

OP No./IP No.: OCCUPATION:

WARD NO.: INCOME:

BED NO.: NATIONALITY:

NAME: RELIGION:

AGE : D.O.A.:

SEX : D.O.D.:

PERMANENT DIAGNOSIS:

ADDRESS:

TEMPORARY ADDRESS:

MEDICAL OFFICER

COMPLAINTS AND DURATION

HISTORY OF PRESENT ILLNESS

HISTORY OF PAST ILLNESS

PERSONAL HISTORY:

a.Food veg/non-veg

b.Marital Statussingle/married

FAMILY HISTORY:

GENERAL EXAMINATION :

Physical built: leannormal obese

Body weight:

Temperature:

Pulse rate:

Heart rate:

Respiratory rate:

Blood Pressure:

Pallor:

Cyanosis:

Jaundice:

Clubbing:

Pedal Oedema:

Lymph adenopathy:

JVP:

Examinations of vital organs:

Heart :

Lungs :

Abdominal organs

Palpation : renal angle

Tenderness Present/Absent

SIDDHA ASPECTS

Yakkai (udalnilai) Mukkunam

1. Vatham 1.Sathuva Gunam
2.Pitham 2.Raasatha Gunam
3.Kapham 3.Thamo Gunam
4.Kalapu

Paruva Kaalam (seasons) Nilam (Places)

1.Kaar Kaalam (Aavani-Puratasi) 1.Kurinchi (Hill area)
2.Koothir Kaalam (Aypasi-Karthigai) 2.Mullai (Forest area)
3.Munpani Kaalam (Maargazhi-Thai) 3.Marutham (Fertile area)
4.Pinpani Kaalam (Maasi-Panguni) 4.Neithal (Sea area)
5.Elavenil Kaalam (Chithirai-Vaikasi) 5.Paalai (Desert area)
6.Mudhuvenil Kaalam (Aani-Aadi)

Iyamporigal/Pulangal Kanmenthiriyam/Kanmavidayam

1.Mei (Sensation) 1.Kai (Koduthal)
2.Vaai (Taste) 2.Kaal (Nadathal)
3.Kann (Vision) 3.Vaai (Pesal)
4.Mooku (Smell) 4.Eruvaai (Kazhithal)
5.Sevi (Hearing) 5.Karuvaai (Aananthithal)

Mummalam

1. Malam
2. Moothiram
3. Viyarvai

Uyir Thathukkal:

Vaatham

1. Pranan 6.Naagan
2. Abanan 7.Koorman
3. Viyanan 8.Kirukaran
4. Udhanan 9.Devadathan
5. Samanan 10.Dhananjayan

Pitham Kapham

1. Analagam	1. Avalambagam
2. Ranjagam	2. Kledagam
3. Saadhagam	3. Podhagam
4. Aalosagam	4. Tharpagam
5. Prasagam	5. Santhigam

Udal Thathukkal

1. Saaram
2. Senneer
3. Oon
4. Kozhuppu
5. Enbu
6. Moolai
7. Sukkilam / Suronitham

Envagai Thervu

1. Naa
2. Niram
3. Mozhi

4. Vizhi
5. Sparisam
6. Malam
   a)Niram
   b)Nurai
   c)Erugal
   d)Elagal

7. Moothiram

1. Neerkuri
   a)Niram
   b)Edai
   c)Manam
   d)Nurai
   e)Enjal
2. Neikuri

8. Naadi

SIGNS AND SYMPTOMS OF KALLADAIPU

Assessment	**Before Treatment**	**After Treatment**			
		10th day	**20th day**	**30th day**	**40th day**
1. Pain ✓ Site ✓ Radiation ✓ Character					
2. Nausea					
3. Vomiting					
4. Burning Micturition					
5. Dysuria					
6. Oliguria					
7. Haematuria					
8. Retention					
9. Fever					
10.Frequency of Micturition					

LABORATORY INVESTIGATIONS

BT **AT**

1. Blooc Tc

Dc

ESR

Hb

B1-Sugar ®

B1-Urea

Sr. Cholesterol

Sr. Creatinine

2. Urine

Colour

Turbidity

Alb

Sug

Dep

- Epethelial cells
- RBC's
- Pus Cells

Casts

Specific gravity

Urine culture and sensitivity if necessary

3. X-RayKUB

4. USG Abdomen & Pelvis

TRIAL DRUGS

Dose:

Anubanam:

Duration of treatment:

Pathiam (Do's and Don'ts):

Prognosis at the end of the treatment

Medical Officer Signature **H.O.D.**

The Tamil Nadu Dr. M.G.R. Medical University

69, Anna Salai, Guindy, Chennai-600 032

This Certificate is awarded to Dr R. SATHYAVATHY

for participating as a ~~Resource Person~~ / Delegate in the VI Workshop on

"Research Methodology & Biostatistics"

for AYUSH Post-Graduates & Researchers

organized by the Department of Siddha

The Tamil Nadu Dr. M.G.R. Medical University

from 12th September 2011 to 16th September 2011

Dr. MAYILVAHANAN NATARAJAN
M.S.Orth. M.Ch.Orth. (L'pool) Ph.D. D.Sc. F.R.C.S. D.Sc. (Hon)^3
VICE CHANCELLOR

Dr. SUDHA SESHAYYAN, M.S.
REGISTRAR (FAC)

Dr. N. KABILAN, M.D. (Siddha)
READER, DEPT. OF SIDDHA

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